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A REDEDICATION?*

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IT has been said that presidential addresses are either a scattering of the philosophical pearls of dotage or the indecent exposure of one's mind. I trust this address may fall into the latter rather than the former category. Perhaps it would therefore not be considered too indecent to give the background of that mind.

Being among the youngest of our generation, we were at each stage perhaps the more impressionable. From a somewhat distracted medical education during World War I, we have possibly never recovered. And then came the first post-war depression. During that period we were exposed to a postgraduate training in a thoroughly co-operative medical atmosphere, from the effects of which we have also possibly never recovered. Then, in a background of an economic boom and a severe depression began an exposure, small in scale but therefore tremendously intimate in detail, to the problems of rural medical service and rural hospitals, medical teaching and medical-school administration, as well as to involvement in the group organization of the staff of a rural hospital attempting to meet the modern demand for an expanding medical service; and then a continuing depression and another world war, which brings us to the present stage of its aftermath. A sampling of presidential addresses should convince one that medicine has been forever at a "crossroads." But our generation has been wandering in a complicated traffic cloverleaf with signposts eliminated one by one.

Against the ever-changing backdrop of this entire period have been demonstrated the marked shifts in public understanding of the economic and sociologic aspects of life in general. This has seen a remarkable scientific development, an application of intensive technologic developments to industry, another series of changes in the concept of the Bill of Rights and, as applied to us and most important of all, a public concept of medical care as a *commodity* rather than a *service*. As a generation we bridge back to the awareness of the public standing and prestige of our predecessors, and we have watched that prestige gradually decline to the point of its lowest ebb. Our lay friends tell us that medicine has lost its soul. This is not a peculiar failure of ours. To the extent that it is true, it has been caught in the sweep of general thinking, influenced by a materialism, an ultra-scientific motif, and an objectivity leading to a belief in nothing except as provable by mathematical, scientific formulas. Endless factors, with which we are all familiar, are responsible. And bouncing across the ceiling as in a nightmare are such words as "the American way of life," "freedom," "freedom of speech," "freedom of press," "freedom of choice" and "free enterprise." And did we mention the term "public relations"? How terribly, as Raymond Fosdick has written, do we need a new vocabulary.

In the latter part of this era American medicine has met a superbly organized, cleverly executed, extensively and subtly financed onslaught with a courageous, necessarily rough and improvised resistance. And now we are resting on our defensive laurels. We seem to have no strategy of offense for again meeting the issues of the battle we have been through. There is little social profit or individual satisfaction in the negative, in the defensive, in the sedentary. We have witnessed the evolution in this era of the acceptance of public responsibility for old age and housing — now health joins the category and requires that we take action to meet our responsibilities or be brushed aside. The question of the socialization of medicine is far from closed. A potential advance in this direction is now apparent in the implications of the program of the Veterans Administration regarding eligibility for hospital and medical care. And in this phase the opposition, a former ally, is of great size and strength. This can affect substantially the adult male population of the country. If we are to be effective participants in the determination of this program, public confidence in the honesty of our position is indispensable. The reorganization of our position requires a cold analysis of the back-

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ground of the issues, recognition of errors of past experience, a realistic development of strategy and a courageous execution of tactics. This is bound to happen if a sufficiently large number of us desire to maintain our autonomy and participate as a force in the evolution of medical service. No profession or occupation has enjoyed the complete freedom of the medical profession — we guard it jealously; do we guard it wisely? We determine who enters the profession, how many, who practices medicine and surgery and how he is trained, and we have carried the responsibility for protecting the public interests involved. Such freedom is founded on confidence and faith. These responsibilities have over the decades been self-imposed, implying freedom of action, and we have insisted that they remain so. Such a state can exist only so long as we fulfill the responsibilities that go with that freedom.

It would be absurd to oppose the concept that access to good health is among the rights of the citizenry. But as a "right" we have the obligation (to ourselves and the American public) to see to it that medical service is placed in its proper position in the order of the necessities and the privileges of life. It is important that we separate the increasing interest of the layman in what he considers his problem of obtaining medical service from the political use or perversion of that interest. If we fail to do this, having now countered the attempt at the political capitalization of that interest, we shall not recognize our shortcomings in the next or present phase of the problem. The attitude of the intelligent layman of today reminds one of Clemenceau's remark during the negotiations after World War I, "war is too important to be left only to the generals." And is it necessary, before such a group as this, to labor through a repetition of the logic involved in the statement that this layman's interest has developed largely as an economic problem of availability of medical service from the standpoint of both access and costs?

Access is available personnel, which he thinks is in short supply. For us to answer that it is not short supply, but unsatisfactory distribution, is merely begging the question — an irritating introduction to a discussion with intelligent men, the majority of whom are willing to make a fair-minded approach to what they consider a public problem. A sufficient supply would soon correct the faulty distribution. We are already seeing the beginning of this development in the recently increased production of general surgeons from a rapid postwar expansion of surgical residencies. Our next counter has been to point out that an increase in the number of doctors has paralleled the increase in the general population — ignoring his implied question, "Were there ever enough?" Our answer is predicated on the false assumption that the demand for medical service has remained static in this period of increased appreciation of its value. He may remind us of public demands in transportation and housing facilities, equipment and so on. Finally, perhaps in desperation, we take recourse in the fact, with implied assumption of total credit, that this country has the greatest health record in the world today. He may quietly suggest that it also has the highest standards of living, the most advanced sanitation, the greatest food availability and the best housing, and he may proceed to the suggestion that we are staking our claims of medical accomplishment rather widely. Or, granting the accomplishment, he may gently suggest that it should not exclude the possibility of improvement.

A vital point of present contact between ourselves and laymen consists in hospital boards of trustees, usually a group of high caliber of intelligence and sincerity. Through increasing utilization of hospitals, increasing costs and deficits, and increasing demands for equipment by professional staffs, these men are being acutely alerted to their responsibilities. Again, the critical state of the economic factors involved raises increasing inquiry into the reasons for so many medical practices and board and staff relations. These laymen learn rapidly that the difficulty of the field in which they are performing requires complete and dependable co-operation from their professional staffs to solve problems that might seem relatively simple within their own spheres of activities. But, in general, little help has been given by us as professional men until it has reached the point where professional staffs are being by-passed in the consideration of the economic problems of medical service in hospitals — wherein most of those problems lie. This is largely due to nonperformance in the past on our own part resulting from a pose adopted, as the late Dr. Frank H. Lahey has well put it, "on our insistence that we are not business men." And the trustees are frustrated by the too often complete absence of staff co-operation and any hope of common policy or attitude within a given staff itself. We have seen the recent deluge of the problems of "hospitals in the practice of medicine" by the full-time employment of professional personnel, and so on. It is again a hopeless confusion of ethics and economics. A reasonably free area of experimentation in the field without the interference of specialty organizations might contribute valuable experience and possible solutions. Again, we might recognize the right of others to an opportunity for some free enterprise. We have not yet read a statement of the radiologic organizations, for instance, that the private patients of a given hospital staff were privately seeking the services of a particular radiologist.

The frustrating position of conscientious boards of trustees or administrators regarding the control of the surgical practice within their institutions

is a veritable nightmare. Because of inability to get positive help from the professional side of the problem, these frustrations can well explode into some other manifestations. Discussions of such items as these within the profession itself again get into a helpless morass of confusion between ethics and economics and between magnanimity and self-interest. Too seldom do we demonstrate a willingness to accept the onus attached to our privileges. Too often do we turn away to preserve the quietude of our intraprofessional relations. Medical economics may change with conditions, but there are some things in this world that to some of us are fixed truths and principles. One of these is the Golden Rule. Others may express it in a more Scriptural manner, but historically a system of principles under the name of "medical ethics" was established for the good of the patient, and indirectly involved relations between physicians. A perverted concept is that it was devised purely for the protection of physicians. In discussion with interns and residents, we have liked to stress the point that the best possible relation between physicians is a primary essential to good medical service to the patient, and also that most of us like to live in peace — in rare instances we have been surprised by the wilful distortions to which this could be subjected.

Never having practiced in an area involved in the system of fee splitting, I have a perhaps naive reaction to its recent and wide publicity. The American public familiar with the system of "kick backs" in many commercial fields and aware of legislative attempts at their elimination, must be thoroughly shocked by the recent and widespread publicity of its performance in the field of medicine. In spite of local courageous attempts that have been made at its elimination, the recent rulings of the Internal Revenue Department and the accompanying publicity of conflicts between state laws and medical-society regulations in this field will certainly leave no possibility of the skeleton's remaining in the closet — and then we wonder about our prestige.

Unnecessary surgery, too, is now a subject of popular magazine articles. The question of the exorbitant fee is no longer a matter of private professional discussion, but to all practical purposes that will soon take care of itself. At the other extreme, the trend toward leveling of fees in general as the result of insurance and compensation schedules is unfair to the extent that it fails to recognize quality — another evidence of this era as the Age of the Common Denominator. The extending breadth of insurance coverage at the same time hardens the traditional atmosphere of medical service in that it will essentially eliminate charity. This has already affected the field of postgraduate surgical teaching as is being elaborated in a committee report to be presented before this meeting. But we had best avoid any further pursuit of this point, which might involve us in a discussion of the philosophic implications of the question, "Why should there be charity?"

So far we have touched on the protection of the interests of the public — a word on our responsibilities from the angle of the supply and training of practitioners. During another lull in the fishing, our lay friend poses the question: "Why does it take thirteen years to produce a medical attendant who can in the initial stage of my illness or accident tell me whether or not I have anything serious requiring more intensive investigation?" Of course, this is an oversimplification of the problem. But after eighteen years of small scale contact and attendance at meetings of medical deans (during which period the personnel turnover was approximately three times), we stand in no awe of the professional educator but in great sympathy for his personnel problems. Medical education in its manner of handling increased volume of material and its pedagogical pattern has been almost completely frozen for many years. With rare exceptions has the present-day medical curriculum been other than the accumulation of increasing scientific knowledge as the piling of layer after layer on a towering cake, the basic layers of which have never been re-examined or re-evaluated for reorganization of the structure or the volume. This extends back even into the scientific attitude of those involved in determining the prerequisite schedule of courses in the premedical program. The vested interest of the specialized scientist has controlled the pattern of the pedagogical program — formally known as a "curriculum." Only our national prosperity has permitted the wastage of time involved, but again the economics of the situation may come to the point of exerting its force. Perhaps the philosophy of a liberal-arts education forces the assumption that the entire content of the process cannot be contained within a lesser number of years. Certainly, there are many misgivings about the present liberal education of the premedical student. But there should be no objection to a liberal education in the proper presentation of a biographical course of outstanding men through medical history presented as focal points in a study of the eras in which they lived — allowing an earlier placement in the program of broad concepts and a background more appreciative of the subsequent more particular material.

The emphasis on the factor of high scientific aptitude in the selection of students should be less absolute. It may well be the mechanism dividing men into those interested in people and those interested in things. So long as the opportunities for men primarily of research interest remain as in the past largely in connection with undergraduate medical schools, why should there not naturally be a lessened interest in the teaching of fundamentals to undergraduate medical students? This situation

has nurtured the concept or the insistence that the undergraduate medical student is a graduate student. Assuming the term "graduate student" to indicate one who has mastered a wide base from which he is to proceed intensively into a narrow sector on his own resources, there is little to support the analogy. And there is the influence of the investigator on the curricular order. I recollect an enthusiast who insisted on introducing the study of pathology with a course in experimental pathology — all of which raises the question of how much of a lifetime is necessary for the basic scientific training and at what point in an educational system such as medicine can a beginning be made this side of the Book of Genesis. What agency is to direct the investigation or restudy of the accumulated material in the curriculum to be covered in the hope of a kind of revised edition that could lead to increased efficiency of time and to some elasticity? Although little progress on this general problem has been made by strictly educational groups, it is interesting that the broader concepts involved in the attack are being urged by clinicians — for example, the recent reports of a committee of the American Surgical Society.

There are many persons who would look with favor on an escalator system, sound at each floor level, that would permit exit at different levels of activity at which different men might wish to emerge. It is not true that every student must go to the top level in training — neither the public needs, individual desires, nor the economics involved will permit it indefinitely — but woe to the scholastic reputation of the institution with the courage to lead the way!

This unappraised mountain of scientific material to be scaled has increased to the point of neglect of the less objective aspects of the patient — his mental workings, environment and personality — that were previously well covered, though perhaps only because there was time for it. The defect was recognized, and the correction unfortunately followed the usual line of procedure, the creation of another special department — in this case (and we remember the period well), the development of psychiatry and its demand for curricular hours. This is not to decry the importance of the human aspect of our job; it is quite the opposite. The present apparent shortage of psychiatrists is but proof of the lack of broadly trained doctors.

An important item in medical service and in its cost and availability is the nurse. We have either neglected the field of nursing education or have been well shoved aside — probably both. An interesting observation on nurse education, through watching this development in terms of the requirements of public service, is that it has repeated every advance and, one might say every error of medical education, the difference being one of time — approximately thirty years later in each instance. Again science has far outrun the emphasis of women on the care of the sick. Here a scientific education for its own sake seems to be too paramount. And we see development of a complete change in the technic of institutional nursing, the justification again being shortage of supply. Division of labor is now introduced to the point that each general nurse on a hospital floor has specialized performance of one small maneuver over the entire floor. We are impressed with the effect of this on the average patient — he realizes that he has ten or twelve nurses or attendants or maids, all of which leads to his conviction that he has none. Medical specialization has resulted in a certain fragmentation of the patient's medical management — we had hoped that the nursing services would provide that one guarantee of the human need of intimate attachment and security, but now they have followed us!

In conclusion, the point should be made that American medicine can maintain its freedom only by the fulfillment of the obligations attached thereto. Can American medicine police itself? We wonder. One may continue to wonder until such organizations as the New England Surgical Society, as well as other components of the grass roots of American medicine, join in giving the stimulation to the leaders of our national organization, which seems to operate on the expectation that its policies and attitudes will be stimulated by local organizations that finally make up the whole. This requires a contribution of time and energy. Groups or movements extending over centuries, and we represent such a group, require occasional reappraisal and rededication to original purposes and traditions. A rededication is due.

Finally, we are in danger of being content to live on a diminishing social prestige built by our predecessors to a proud place through self-sacrifice and warm, human, individual responsibility. We are in danger of discovering that we have less inner satisfaction for ourselves and less pride of place to pass on to our successors. Our profession creates its eminence through deeds, not words, and through meeting human needs, not in satisfying our own. The correlation is clear; the danger is not remote.

UNRECOGNIZED EMBOLI TO THE LUNGS WITH SUBSEQUENT COR PULMONALE*

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IN recent years interest in pulmonary embolization has centered largely about preventive measures, with emphasis on leg-vein ligation and anticoagulant therapy.[1, 2] The principal cause for concern has been the acute syndrome of pulmonary infarction with its all too familiar picture of chest pain, hemoptysis and sometimes sudden death. It is well known, however, that pulmonary emboli may occur without clinical recognition,[3-5] and the incidental postmortem finding of organized emboli involving scattered vessels is not uncommon.

There is a special group of patients, however, in whom the emboli are so extensive that they ultimately result in serious obstruction to the flow of blood in the lungs. This puts a burden on the right ventricle that eventually leads to cor pulmonale. We have seen 12 examples of this sequence uncomplicated by other significant disease at the Massachusetts General Hospital among 8000 autopsies during the past twenty years. These patients usually present the clinical picture of increasingly severe congestive heart failure of undetermined cause. Often they have been considered cases of mitral stenosis "without a murmur" or some unusual form of congenital heart disease. It is only in recent years with cumulative experience and autopsy confirmation that this syndrome has been fully appreciated and the correct diagnosis suspected during life.

This report includes only cases with extensive old pulmonary embolization and hypertrophy of the right ventricle without other complicating diseases that might have contributed to the cardiorespiratory impairment.[1, 6-9] The accumulation of only 12 cases in twenty years suggests that this is a rare condition. However, these figures are somewhat misleading, since we purposely selected only the patients with the syndrome in its "purest" form in order to emphasize this entity. Therefore, many cases in which pulmonary embolization contributed to a disability produced in part by other diseases have been excluded.

The principal clinical and pathological aspects of these 12 cases are shown in Table 1 and in Figures 1-5. We have selected the following 5 cases to emphasize the typical features of this syndrome:

CASE 2. A 41-year-old hardware jobber was first seen in 1952. He had been well until 4 months previously, when he first noted a nonproductive cough. A month later swelling of the ankles and marked dyspnea on exertion appeared, and he soon needed 2 or 3 pillows at night. His physician prescribed digitalis and a low-sodium diet, with little benefit. He had coughed up half a teaspoonful of blood on three occasions in recent weeks. He had not had chest pain and denied previous hypertension or rheumatic fever.

Physical examination revealed a slightly cyanotic man with distended neck veins in the upright position. There were moderate cardiac enlargement, sinus tachycardia (rate of 110 per minute) and a blood pressure of 120/80. The pulmonic 2d sound was accentuated, and there was a slightly harsh, Grade 2, systolic murmur heard best along the left sternal border. The liver edge was palpable 2 fingerbreadths below the right costal margin, and there were signs of ascites. A +++ pitting edema of the ankles was present, but no calf tenderness was noted.

Examination of the blood showed a hemoglobin of 12.2 gm. per 100 cc., a carbon dioxide of 28.5 milliequiv. and a sodium of 133 milliequiv. per liter, and a pH of 7.46. The vital capacity was 1.4 liters. The blood Hinton test was negative. An electrocardiogram demonstrated the characteristic pattern of right-ventricle hypertrophy (Fig. 1). A roentgenogram of the chest revealed clear lungs except for an unusual "strand-like" density in the left upper lobe, a prominent pulmonary-artery shadow and an increased transverse diameter of the heart. At cardiac catheterization the pressure in the right pulmonary artery was equivalent to 100-116 mm. of mercury systolic and 34-42 mm. diastolic, with a mean of 62 mm. Pressure in the right ventricle was 116 mm. systolic and 4-20 mm. diastolic. Arterial oxygen saturation was 92 per cent at rest. These findings were consistent with cor pulmonale and failure of the right ventricle. The patient was discharged on a regimen of digitalis, sodium restriction, mercurial diuretics and codeine for cough. He continued to do poorly at home, with increasingly severe dyspnea and cough, and died 3½ months later — 7½ months after the onset of the initial symptoms.

At post-mortem examination the heart demonstrated marked hypertrophy and dilatation of the right ventricle, with a relatively normal left ventricle. The heart weighed 480 gm. The right-ventricle wall was 0.9 cm. thick (approximately three times the normal), and the left 1.0 cm. thick (low normal). No other cardiac abnormalities were noted. The lungs were only slightly increased in weight (950 gm.) and were crepitant throughout, with minimal terminal edema. The only findings of significance were in the pulmonary arteries. Gross examination showed the branch to the left lower lobe to be completely occluded by an old fibrosed embolus. Similar fibrous material was seen in the branch to the left upper lobe, with, in addition, some fresher, red-brown emboli. Similar old and recent emboli were seen in smaller branches of the right pulmonary artery. Histologic examination of sections from all lobes disclosed organized recanalized emboli in the branches of the pulmonary-artery tree at all levels. These were particularly numerous in the small arteries and arterioles. In some sections almost every vessel contained thrombotic material. Dissection of the leg veins showed extensive old and recent thrombosis of the right common femoral vein. Ascites and a right hydrothorax were present.

This case is characteristic of the group, in that cor pulmonale developed slowly and insidiously as a result of increasing pulmonary hypertension from widespread recurrent emboli to the smaller lung

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vessels without recognizable episodes of pulmonary embolization. Although in retrospect it is likely that the hemoptyses represented small infarcts, the virtually clear chest roentgenograms and the lack of chest pain or calf tenderness caused confusion regarding the true nature of the illness.

CASE 6. A 65-year-old unemployed man entered the hospital with acute urinary retention. For some months he had noted the lung fields remained clear. At one point a suspicious density was noted in the right mid-lung field on x-ray examination, but its exact nature was not clear. He died 5 weeks after admission in congestive heart failure.

Clinical diagnoses were carcinoma of the prostate, retroperitoneal sepsis, multiple pulmonary infarcts and congestive heart failure. The post-mortem findings are presented in Table 1.

CASE 8. A 52-year-old Negro housewife entered the hospital complaining of cough and dyspnea for 1 year. Eighteen years previously she had had "pneumonia and influenza." Two or

TABLE 1. *Summary of Clinical and Pathological Data.**

CASE NO.	SEX	AGE	DURATION OF SYMPTOMS	CYANOSIS	HEMOPTYSIS	MURMURS ALONG LEFT STERNAL BORDER	ASCITES	CLUBBING	ARTERIAL OXYGEN SATURATION	HEMOGLOBIN	CARBON DIOXIDE CONTENT
		yr.							%	gm./100 cc.	milliequiv./liter
1	M	32	10 yr.	+	+	+	+	0	—	14.5	18.1
2	M	41	7½ mo.	+	+	+	+	0	92.0	12.2	28.5
3	M	72	17 yr.	+	0	0	+	0	—	(80%)	—
4	F	49	6 wk.	+	+	0	0	+	—	11.5	—
5	M	55	10 mo.	+	0	0	0	0	—	13.6	—
6	M	65	"Few wk."	0	0	0	0	0	—	(80%)	—
7	F	53	1½ yr.	0	0	0	0	0	—	16.0	—
8	F	52	1½ yr.	+	+	+	0	0	—	14.0	25.4
9	F	48	10 mo.	+	+	+	0	+	—	17.0	32
10	F	44	7½ yr.	+	0	0	+	0	93.4	(88%)	22.9
11	F	32	7½ mo.	+	0	0	+	0	92.0	16.4	—
12	F	47	14 mo.	+	0	+	+	0	—	16.0	—

*Findings in following cases previously published: Case 1,[10] Case 2,[11] Case 3,[12] Case 5,[13] Case 7,[14] Case 8,[15] Case 9,[16] Case 10,[17] Case 11[18] and Case 12.[19]

symptoms of prostatism. A few weeks before, he had had two episodes of "dizziness," and for a week he had felt "chilly." He gave no history of heart disease and denied dyspnea on exertion, orthopnea or ankle edema. The blood pressure was 130/80. The pulse was regular at a rate of 95 per minute.

On physical examination the heart was not enlarged, and the lung fields were clear to auscultation. The pulmonic 2d sound was louder than the aortic, and a Grade 2 systolic murmur was heard at the base of the heart.

One week after admission a perineal prostatectomy was done, and carcinoma of the prostate with local extension was found.

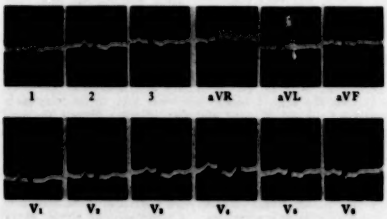

FIGURE 1. *Electrocardiogram in Case 2, Showing the Typical Pattern of Hypertrophy of the Right Ventricle.*

Nine days later an episode of dizziness, dyspnea and hypotension occurred. After this the patient noted increasingly severe dyspnea on exertion and soon began to lose responsiveness. His neck veins were pulsating in the upright position, the pulmonic 2d sound was louder, and a diastolic gallop rhythm had appeared. The heart was now enlarged on percussion, though

three spells of sharp precordial pain lasting only a few moments but with exacerbation on exertion had occurred each year for the past 5 or 6 years. Twelve months before entry she again had "pneumonia," with chills and cough, and dyspnea on exertion and a chronic cough subsequently appeared. Four months later she had a third bout of "pneumonia" with cough, pleurisy

FIGURE 2. *Hemisection of the Left Lung in Case 3, Showing the Left Main Pulmonary Artery Filled with a Large, Old Embolus.*

and "rusty" sputum. After this she complained of increasing dyspnea on exertion, orthopnea and ankle edema. She had frequent paroxysms of coughing, at times productive of bright-red blood. Her legs became swollen, especially the right, and by the time of admission she could sleep only in the upright position in a chair.

Physical examination showed an orthopneic woman who appeared seriously ill. The heart was greatly enlarged, with a loud pulmonic 2d sound. A harsh systolic murmur was heard along the left sternal border, and a few inspiratory rales were present at both lung bases. The blood pressure was 110/82. There was regular rhythm at 90 per minute. The liver edge was palpated 2 fingerbreadths below the right costal margin,

and there was a ++ to +++ pitting edema of the lower legs.

X-ray examination of the chest revealed enlargement of the right ventricle and prominence of the pulmonary artery. The lung fields themselves were clear — possibly more so than normally. An electrocardiogram demonstrated right-axis deviation. The blood Hinton test was negative. The red-cell count was 4,050,000, with a hemoglobin of 14 gm. per 100 cc.

The patient was treated with digitalis and diuretics and lost much of the excess fluid. She improved symptomatically over a period of several weeks and was then discharged home, but after 8 days returned to the hospital complaining of epigastric pain and increased shortness of breath. She was now very cyanotic and orthopneic and died suddenly a few hours after arrival.

Clinical diagnoses were coronary-artery disease with congestive heart failure and chronic nephrosclerosis. Table 1 presents the post-mortem findings.

TABLE 1 (*Concluded*).

Case No.	Electrocardiographic Findings	Pulmonary Emboli: Main Pulmonary Arteries	Pulmonary Emboli: Small Arteries	Source of Emboli	Small Pulmonary Infarcts	Heart Weight	Thickness of Right Ventricle
						gm.	mm.
1	Hypertrophy of right ventricle	At bifurcation completely occluding right & 50% of left branch	++ ++	Left femoral vein	0	610	10
2	Hypertrophy of right ventricle	Left-lower-lobe branch completely filled & left-upper-lobe branch partially filled	++ +	Right femoral vein	0	480	9
3	Right-axis deviation	Left completely & right partly occluded	+	Popliteal & iliac veins & vena cava	0	525	8
4	Left-axis deviation	Large recent embolus in main artery; many organized emboli in small vessels.	++ +	Both femoral veins	0	390	6
5	Right-axis deviation	Both main branches almost completely occluded	++ ++	Popliteal veins	++	400	7
6	?	None in main arteries, but almost all of medium size obstructed	++ ++	Right femoral vein	++	520	5-6
7	Hypertrophy of right ventricle	None in large arteries	++	Portacaval shunt (see text)	0	300	7
8	Right-axis deviation	Both main branches almost completely occluded	++ +	Right atrial appendage	+	330	6-8
9	Hypertrophy of right ventricle	Right-lower-lobe branch filled	−− −−	Heart (tumor thrombus, right ventricle)	++	350	5-15
10	Right-axis deviation	None in main (but 2/3 of the way to pleura, numerous)	+++ ++	? (legs not examined)	++	520	6-10
11	Right-axis deviation	None in main arteries	++	? (legs not examined)	++	400	10
12	Hypertrophy of right ventricle	Both right & left main partially occluded	−−	? (none found)	0	300	7

Case 9. A 48-year-old woman was admitted to the hospital because of exertional dyspnea and chronic cough. Six years previously she had been treated with radium for carcinoma of the cervix. A pelvic examination 3 months previously had revealed no recurrence. Her health had been otherwise good until 9 months before entry, when she was in an automobile accident and was thrown out of the vehicle onto the street. Though no serious injuries were detected at this time, she soon noted dyspnea after climbing one flight of stairs, and the onset of a chronic cough. Two weeks after the accident, there was an attack of pain in the right anterior side of the chest that was exacerbated by coughing. She had several similar episodes of chest pain in subsequent months, along with persistent shortness of breath and cough. Three months before admission she began to have hemoptysis, which consisted of blood streaking of yellowish sputum and occurred several times each week. A "spot" on the right lung was noted on x-ray examination at this time, clearing in a day or so. Bronchoscopic examination was negative. During the past month she had felt very ill and had noted an increase in dyspnea. She could sleep only with the support of 3 pillows, and her ankles became swollen late in the day.

Physical examination showed a woman who appeared chronically ill and had a cyanotic tinge to the lips. The neck veins were distended, and the heart was considerably enlarged to percussion. A loud diastolic gallop rhythm was audible along the left sternal border, and in the same area a harsh systolic murmur was noted. The lungs were clear to auscultation. The liver edge was felt 4 fingerbreadths below the right costal margin, and there was slight swelling of the ankles. An electrocardiogram disclosed the characteristic pattern of right-ventricle hypertrophy. Chest films revealed densities in the right upper lobe and left lower lobe consistent with pulmonary infarction; these cleared under observation in the hospital. The hematocrit was 53 and 50 per cent on 2 determinations. The patient was treated with a program of digitalis, ammonium chloride, mercurial diuretics and sodium restriction. On this regime she lost 8½ pounds in 16 days. Because pulmonary embolism was suspected, she was also maintained on Dicumarol therapy from the day of admission. During hospitalization she complained repeatedly of pleuritic pain in various parts of the chest. She had a chronic cough, with blood-tinged sputum much of the time. Despite some diuresis her symptoms of dyspnea and cough were little improved, and she died about 4 weeks after admission.

The clinical diagnosis was cor pulmonale secondary to multiple pulmonary emboli. Table 1 presents the post-mortem findings.

Case 12. A 47-year-old housewife was admitted to the hospital because of increasing shortness of breath and cough. Three pregnancies (the most recent 17 years before) had been uneventful. One year before entry fatigue, dyspnea, edema of the face and legs, abdominal swelling and slight cyanosis of the lips gradually appeared. Her physician treated her with injections of mercurial diuretic twice weekly, ammonium chloride, a low-sodium diet and digitalis tablets during a 3-month period of bed rest. On this regimen she improved, with loss of the edema. However, the symptoms soon recurred, and diuretic measures proved unsuccessful.

Physical examination showed a well developed but thin woman who appeared chronically ill. There was slight cyanosis of the lips. The superficial and deep neck veins were distended and pulsating. The left border of the heart was in the anterior axillary line, with a diffuse apical impulse. There was a very loud, harsh systolic murmur, heard best along the left sternal border. It was accompanied by a systolic thrill. There was slight sinus tachycardia (rate of 100 to 105 per minute) and a blood pressure of 110/80. The lungs were clear to auscultation. The liver edge was felt 3 or 4 fingerbreadths below the right costal margin and was tender to palpation. There were ascites and pitting ankle edema of both lower legs. An electrocardiogram revealed the pattern of right-ventricle hypertrophy. On fluoroscopic examination the heart shadow was increased, with prominence of the pulmonary-artery shadow and hilar vessels. The radiologist noted that the peripheral lung fields were less vascular than the usual lung fields. The hemoglobin was 16.0 grams per 100 cc., and the vital capacity 2.1 liters (63 per cent of normal).

During 5 weeks in the hospital the patient lost 22½ pounds on digitalis, sodium restriction and diuretics. There was considerable symptomatic improvement, but slight cyanosis, tachy-

cardia and ascites persisted. Three days after discharge she suddenly died at home.

The clinical diagnosis was cor pulmonale, of undetermined etiology.

Clinical Features

Dyspnea and *cough* were the most frequent symptoms encountered in this group of patients. Shortness of breath on exertion was present in every case and was the initial complaint in 10 cases. One patient (Case 8) came to the hospital because of *hoarseness*. She had paralysis of the left vocal cord owing to pressure on the left recurrent laryngeal nerve by a tense, distended pulmonary artery. It is of interest that because of the paralyzed vocal cord and a "mass" at the hilus of the lung, neoplastic disease was suspected, but surgical exploration revealed only a large, pulsating pulmonary artery. This symptom from vocal-cord paralysis has been encountered by others in this syndrome as well as in Eisenmenger's complex, defect of the atrial septum, patent ductus arteriosus and mitral stenosis — all presumably on the same basis.[20,21] *Syncope*, which has been reported by others as a helpful sign in diagnosis of "primary" or "essential" pulmonary hypertension,[22] was encountered only in Case 6. Despite the fact that actual infarction of the lung is not the important factor here, 5 of the 12 patients had at least one episode of *hemoptysis*. In some this recurred.

As a rule, the pulsating neck veins, enlargement of the liver and extensive ankle edema of a failing right ventricle were present late in the course of illness. *Ascites* was noted in 6 cases. These findings emphasize the extreme degree of right-sided failure that ultimately ensues. *Cyanosis* was one of the most constant physical findings, being present in 10 of the 12 cases. An accurate evaluation of this finding was complicated by the presence of heart failure in each patient, since peripheral stasis itself may result in mild cyanosis.[23] However, it is well known that pulmonary vascular disease is a sufficient cause of cyanosis without shunts or heart failure, and cyanosis is helpful in suggesting the syndrome. Though *clubbing* of the fingers and *polycythemia* were encountered, it is interesting that they were the exception and were of slight degree. This can probably be correlated with the minimal degree of arterial oxygen unsaturation observed in these patients and with the absence of pulmonary sepsis.[24] However, Harvey et al.[25] reported a case of this type with data showing considerable hypoxia. Serum carbon dioxide values were near normal levels, as might have been expected in the presence of congestive heart failure[23] and pulmonary ischemia with no significant ventilatory embarrassment.[26] Patients with emphysema or other important pulmonary disease that might have resulted in elevated carbon dioxide levels were excluded from this report.[25, 27]

The pulmonic second sound was invariably accentuated, and the heart enlarged by percussion. Although there were no diagnostic *auscultatory* findings, gallop rhythm was encountered often, and 5 patients had a harsh, Grade 3 to 4 systolic murmur, heard best along the left sternal border in the third or fourth interspace. Post-mortem examination in each of these patients revealed a large organized embolus in the main pulmonary artery or in one of the main branches. Turbulence in a large vessel from such a clot rather than from pulmonary hypertension alone seems a possible basis for these murmurs. However, the same type of clot was noted in other cases without a similar murmur. The *rhythm* was regular in every case, which is the rule in patients with cor pulmonale from any cause.[28]

It was a striking feature that *x-ray* examination offered little positive help in diagnosis in the earlier cases, but a recent review from this hospital[29] calls attention to the suggestive clue of clear lungs in the presence of prominent root vessels. This appearance of relative ischemia of the lung fields was noted in 5 cases in this study. It is in accord with the pathological findings, and its diagnostic importance is worthy of special emphasis. In most cases the fluoroscopist found prominence of the main pulmonary vessels and enlargement of the right ventricle but the rarity of actual lung infarction in this syndrome helps explain the difficulty facing the radiologist. In Case 1 a correct diagnosis of an organized embolus in the major arterial channels of the lung was suspected from the films because of the paucity of lung markings distal to the prominent vessel shadow.

Electrocardiograms were available in 11 of the 12 patients, with evidence of right-ventricle hypertrophy in all but 1. In the 5 cases studied before multiple precordial leads were routinely used the tracings revealed right-axis deviation. Of the 5 cases studied at a later date, with a full set of precordial leads, the characteristic pattern of right-ventricle hypertrophy was observed in all. The remaining tracing showed extreme left-axis deviation (−90°), but this heart showed marked hypertrophy of the right ventricle with no enlargement of the left ventricle at autopsy. This is an unusual and puzzling combination, but it has been encountered by others, and the discordant electrocardiogram can probably be explained by vector methods. The failure to encounter right-bundle-branch block despite long standing hypertrophy of the right ventricle was interesting, in view of the frequent finding of left-bundle-branch block in cases of chronic left-ventricle enlargement.

Pathological Observations

A striking feature in all cases was the marked hypertrophy of the right ventricle, with walls varying from 6 mm. to 15 mm. in thickness (normal, 3 or

4 mm.). In most cases there was also dilatation of the right ventricle and atrium, with relatively normal left-sided chambers. None showed congenital anomalies, valvular lesions or significant coronary-artery disease.

The abnormalities in the lungs were largely limited to the vascular tree. None showed extensive infarction, although small, relatively insignificant infarcts of varying ages were seen in 5 cases. It should be stressed again that infarction is not a prominent feature or a causative factor in this syndrome.

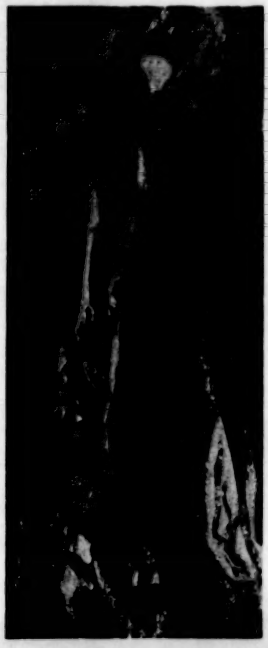

FIGURE 3. *Longitudinal Section of a Medium-Sized Artery in Case 10, Showing a Web-Like Organized Embolus in the Mid-Portion. (Reproduced from Castleman and Bland[29] by Permission of the Publishers.)*

The most striking change and constant finding in the pulmonary-artery tree was the extensive and extreme degree of embolization. The size, age and location of the emboli varied considerably, but in 10 cases one or more of the large branches of the pulmonary artery contained emboli readily demonstrable on gross examination (Fig. 2, 3 and 4). All the 12 cases showed organized emboli in the tertiary branches of both lungs on microscopical examination. In addition, a few cases demonstrated superimposed fresh emboli. When older emboli were present in the main pulmonary artery or in one of the major branches there was invasion of the periphery of the thrombotic material by fibroblasts, the central part having an amorphous appearance.

The organized emboli in the arteries less than a few millimeters in size showed complete replacement of the thrombotic material by collagenous and elastic tissue. This characteristically took the form of a meshwork composed largely of collagen, with varying numbers of newly formed vascular channels often surrounded by delicate elastic fibers (Fig. 5 *A*, *B*, *C* and *D*). Some of the lesions were so well organized that they closely resembled arteriosclerosis (Fig. 5 *E*). This feature has also been noted in experimental embolization in animals.

It is difficult to measure the degree of obstruction caused by these lesions in the aggregate, but in all cases it was considerable, and in no case was it limited to one lobe or even to one lung. The relative

FIGURE 4. *Cross Section at the Level of the Embolus.*

amount of obstruction produced by the emboli in the large vessels and those in the smaller ones varied, but in all cases the emboli in the smaller vessels were a prominent feature. Many vessels not containing emboli at the level of the sections showed prominent fibrous intimal thickening or atheromatous plaques. It was demonstrated clearly in Case 10 (previously reported)[29] that these changes in the intima took place proximal to the area of obstruction. The site of obstruction could be predicted from the appearance of the vessel on cross section. If the wall was thin, the obstruction was proximal to the cut. If it was thick and fibrous, the obstruction was distal, indicating pulmonary hypertension of long standing.

The source of pulmonary emboli was demonstrated in 9 cases; a site was found in the leg veins in 6 and in 2 others in the heart. In Case 7 there was an unusual and probably unique relation involving an

obstruction of the portal vein, with resulting extensive large anastomoses with the vena cava. Many of the anastomotic vessels contained thrombi, and it seems likely that these were the source of the pulmonary emboli.

In 2 of the 3 cases in which no source was demonstrable, dissection of the leg veins was not done, because of either autopsy restrictions or failure to recognize the significance of the lung changes at the time of autopsy.

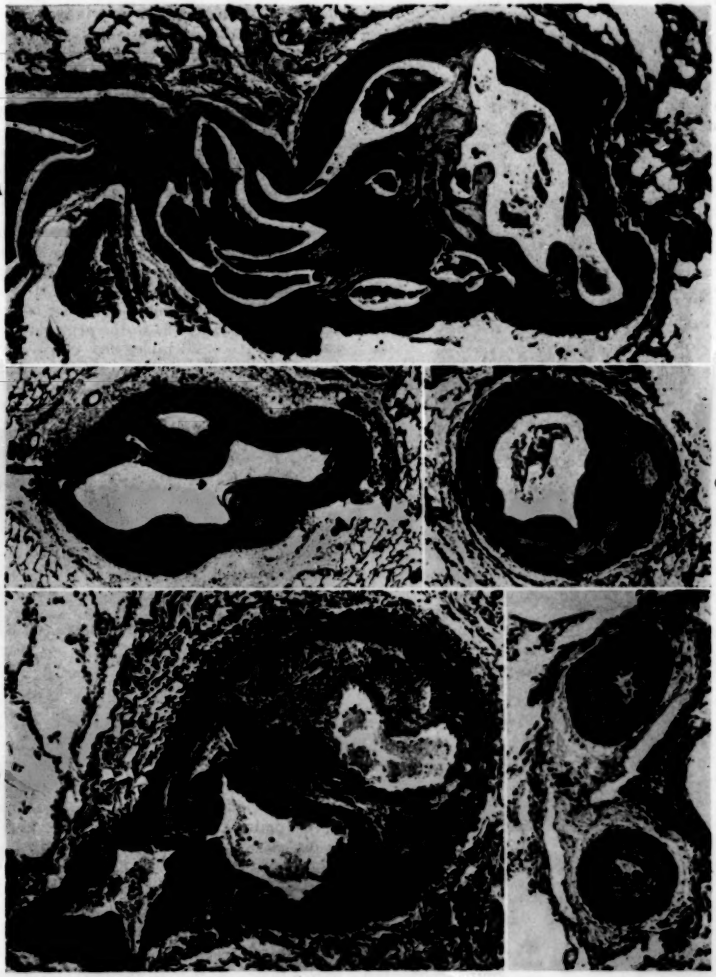

FIGURE 5. *Photomicrographs of Various Sections, Showing Low Magnification of the Characteristic Organized Emboli* (A, B *and* C), *High Magnification of an Organized Embolus of Long Standing, with Subsequent Recanalization* (D) *and Two Small Arteries Revealing Changes Indistinguishable from Arteriosclerosis (This Finding Was Noted in All Cases).*

Discussion

Cases similar in many respects to these have been reported in the literature as primary pulmonary arteriosclerosis, primary pulmonary hypertension or as the ill defined condition called Ayerza's syndrome. We believe that "primary" disease of the pulmonary artery is exceedingly rare and that pulmonary embolization (often dating back many years) should be excluded before such a diagnosis is tenable.

Changes in the pulmonary vessels identical with these have been produced in laboratory animals by the injection of various substances into peripheral veins. These include macerated autogenous[30, 31] and heterogenous[32, 33] blood clots, amniotic fluid[34] and bits of filter paper.[35] The resultant changes in the lungs in the animals varied widely with the quantity of material injected and the frequency of injections. Repeated injections of a quantity of material only slightly less than that required to cause sudden death resulted in multiple organized emboli and fibrous intimal thickening — findings indistinguishable microscopically from localized arteriosclerosis. However, a single injection of blood clot resulted in only temporary changes and, when the animals were killed after a suitable period had elapsed, only scant lesions could be found in the pulmonary vessels.

This suggests that most of our patients had been the victims of repeated episodes in which multiple small emboli were fed to the lungs from the peripheral veins or occasionally from the right chambers of the heart. They may also have occurred when one or two large emboli underwent partial disintegration after lodgment in a major pulmonary vessel. This is borne out by the presence of multiple emboli in the small vessels, even in the cases in which the main pulmonary artery was almost completely filled.

Primary pulmonary thrombosis as the sole factor in some cases or as an additional factor in others cannot be completely excluded. However, this possibility seems unlikely from an anatomic standpoint, since the structural changes are identical in the cases that seem to be unequivocal examples of pulmonary embolization and in those in which the evidence is less conclusive. Moreover, phlebothrombosis in the peripheral veins is extremely common and, if the resulting emboli do not produce sudden death or infarction, they may well pass unnoticed and the associated symptoms of transient dyspnea and possibly cough may be forgotten by the time cor pulmonale becomes evident years later.

Under these circumstances the interval between the obstruction and clinically evident cor pulmonale is difficult to establish and probably quite variable. From the histologic appearance of the emboli and the secondary vessel wall thickenings it seems to be measured in years rather than in weeks or even months.

Those cases with large organized emboli in the main pulmonary artery, in addition to those in the smaller vessels, did not present any clinical features to distinguish them from those with emboli only in the smaller channels. Others have noted that emboli limited to the main channels of the pulmonary artery may result in cor pulmonale.[6]

Summary

Twelve cases of chronic cor pulmonale and heart failure as a result of multiple and, for the most part, "silent" pulmonary emboli are presented.

Post-mortem study revealed the role of widespread, organized embolization of the lungs as the only etiologic factor responsible in these patients. The tertiary branches of the arterial tree were widely obstructed in all cases, and the larger branches in several.

In each patient dyspnea and cough antedated the appearance of hypertrophy of the right ventricle and congestive heart failure. Gallop rhythm was the rule. Frequently cyanosis and a harsh systolic murmur along the left sternal border were present. Clubbing of the fingers, polycythemia and slight arterial oxygen unsaturation were encountered infrequently. All patients succumbed to intractable congestive heart failure. The etiologic diagnosis was usually unsuspected.

This study suggests that a large proportion of cases of so-called Ayerza's syndrome result from latent embolization of the lungs.

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PILONIDAL SINUS*

Experience with 449 Cases

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SINCE the first case was reported in 1847[1] a voluminous literature on the origin and treatment of pilonidal sinus has accumulated. Although some of the earlier authors regarded the condition as acquired[2, 3] the consensus has been that it is congenital. The two prevalent theories are that pilonidal sinuses represent remnants of the neural canal[4-8] and that they are due to an ectodermal inclusion resulting from malfusion of the two halves of the body.[9-11] The proponents of both theories demonstrate a space or canal lying between the coccyx and the skin, which is constant in small embryos[6] and variable in fetuses,[4] and has usually disappeared at birth. A skin dimple, at times deep enough to be called a sinus, is present in 20 to 33 per cent of newborn infants but persists in only 4 or 5 per cent of adults.[6] Mallory[4] believed that the absence of glands and hair follicles in the fetal sinuses is inexplicable if they are an ectodermal inclusion; however, he does not satisfactorily explain the presence of hair if the origin of the sinus is neural. Kooistra[6] voices the opinion of both schools when he says: "It is obvious from the pathology that any form of treatment to be successful must completely eradicate the cyst and sinus and all granulation tissue." This belief has been the basis of most operations for pilonidal sinuses.

TABLE 1. *Results of Various Operations for Pilonidal Sinus.*

OPERATION	PATIENTS OPERATED ON	UNTRACED CASES	PATIENTS FOLLOWED FOR ONE YEAR OR MORE	SEPSIS IN CLOSED OPERATIONS	RECURRENCE	NO RECURRENCE
				NO. OF CASES	NO. OF CASES	NO. OF CASES
Excision & simple closure	27	3	24	1	2	22 (92%)
Excision & partial closure	18	1	17		1	16 (94%)
Excision & gluteal-flap closure	168	9	159	14*	19	131 (82%)
Excision & wire closure	50	2	48	5	6	42 (88%)
Excision & packing	87	12	75		9	66 (88%)
Incision & packing (abscesses)	45	5	40		3	37 (93%)
Incision & packing	54	6	48		6	42 (88%)
Totals	449	38	411	20	46	356 (87%)

*9 of these required reoperation and are classed as failures.

Patients whose wounds healed without reoperation and remained healed for a year are classed as good end results even if there was delayed healing from sepsis.

Our experience with various operations in 449 patients during the past four years has led us to question these concepts of etiology, and to deny the necessity of removing the sinus tract unless the purpose of excision is to allow primary closure, which we use in selected cases.

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A hundred and sixty-eight patients were operated on with the technic of Shute et al.,[12] flaps of gluteal muscle and fascia being employed to close the dead space left by excision of the lesion. A hundred and thirty-five of these cases have been reported.[13] The subsequent 33 cases did not change the statistical results. In 9 per cent of the whole series there was wound sepsis, causing delayed healing or complete failure, and in 12 per cent there was recurrence. This is not an excessively high rate for a closed operation, but the difficulties encountered in the cases regarded as failures, the prolonged healing time and in some cases the need for multiple operations led us to the conclusion that an extensive plastic procedure of this type is not a justifiable method of treating pilonidal sinus.

A second group of 27 patients, all with small, midline, uninflamed lesions, was treated by minimal excision and closure with either buried sutures or deep stay sutures tied over a gauze pack. These cases are included (Table 1, "simple closure") for completeness. They were treated, for the most part, before the gluteal-flap series was begun. There was not sufficient uniformity in the method of closure to warrant any conclusions.

Seventy-five cases, deemed unsuitable for primary closure, were treated by open excision, as described by Rogers and Dwight,[14] except that the actual cautery was rarely used. The principle of minimal excision of the sinus tracts was adhered to, and the wounds were treated by packing until healed. The median healing time was nine weeks, the same as that in the series of Rogers and Dwight,[14] with a range of five to twenty-five weeks.

A modification of this method ("partial closure") was used in 17 cases, the skin edges being sewed down to the sacral fascia. This technic is useful only with a straight, midline lesion, and cannot be employed in obese patients in whom the defects are very deep.

Pilonidal abscesses were incised and drained, with the intention of performing a later excision, with or without primary closure. The incision was always a midline one, regardless of whether the abscess was pointing there or laterally. This was done to facilitate later excision and on the assumption that since the abscess originated from a midline process, it could be drained adequately through a midline incision. A further step was to extend the midline incision to include all midline sinus openings, whether they fell in the inflamed area or not. The entire cavity was loosely packed, rather than kept open by a drain. Packing was continued until the wound healed. It was found that the wound healed rapidly and cleanly, and in the great majority of cases there was no need for further surgery. Forty-five patients were treated in this way. There were 3 recurrences, 2 of them in patients who had had postoperative care elsewhere.

The success of this method led to the next step, incision and packing of the chronic sinuses. No tissue was excised, but all tracts were laid open, nests of hair removed, unhealthy-looking granulation tissue wiped away with gauze, and skin edges trimmed when necessary to avoid overhanging edges or awkward angles where lateral tracts entered the midline incision. The subsequent care was the same as that after excision. There were 54 of these operations with six recurrences in the 48 patients traced. Figure 1 demonstrates the healing time of these cases compared with those treated by excision and packing; the maximum was twelve weeks.

Since we were unwilling to relinquish entirely the quest for a satisfactory method of primary closure, 50 patients were treated by a technic[15] not yet

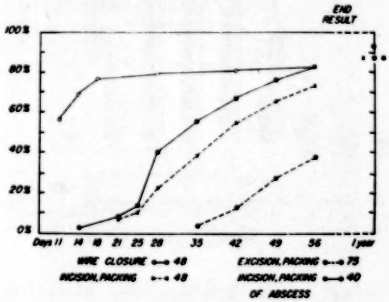

FIGURE 1. *Healing Time, Expressed as Percentage of Cases Treated at Intervals up to Fifty-Six Days after Operation, and End Result One Year from the Time the Wound Was Healed.*

described in the literature. This operation should be used only for midline lesions 8 cm. or less in length and with no extension more than 2 cm. from the midline. The lesion is excised with a minimal amount of skin and fat, including the tract and its openings (Fig. 2*B*), care being taken to stay just outside the cyst. After removal of the lesion the defect is closed with running sutures of No. 000 braided Surgalloy wire. One suture on a large, curved needle is started on the right side entering through the skin about 2 cm. lateral to the cephalad end of the incision. This suture is brought into the upper end of the wound at its deepest level, and the large needle is replaced by a smaller one. It is carried from side to side, taking bites of the fat, somewhat as in a continuous mattress suture (Fig. 2*B-a*). A doubled wire suture (*a'*) twisted to make a very small loop is threaded onto a large cutting needle, introduced at the lower end of the incision and brought out 2 cm. lateral to it. The first suture is threaded through this loop, which serves as a

pull-out wire, and then continued at a slightly more superficial level, back up the wound, and brought out beside its origin (Fig. 2*C*). The two ends are tied over a pledget of gauze, after the second suture has been placed. A second running suture (*b*) is started on the left, goes down the wound to a pull-out loop on the right (*b'*), and comes back as a subcuticular stitch (Fig. 2*D*). In this way the wound is closed in four layers by two running

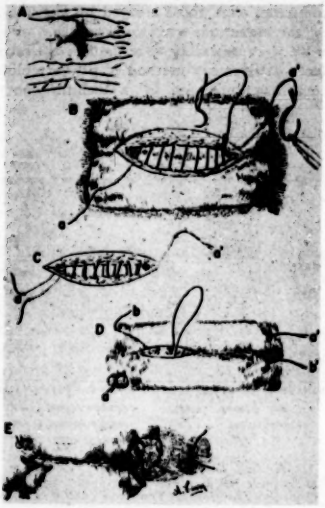

FIGURE 2. *Technic of Wire Closure.*

sutures, each one starting and ending at the upper end and going through a pull-out at the lower (Fig. 2*E*). The ends of the two pull-outs are tied to each other over a roll of gauze. The patients are kept in bed for five days, on 600,000 units of penicillin daily, low-residue diet and deodorized tincture of opium, 10 drops three times a day. On the tenth day the sutures are cut at the upper end and removed by tension on the pull-out wires.

Occasionally, the wire broke and part of it remained. Usually, this caused no inconvenience, but in a few cases one end of the wire was near enough to the skin to cause a pricking sensation, in which case it was easy to find and remove. In 5 cases in which sepsis necessitated early removal of the sutures, the wounds healed in four to twelve weeks without reoperation. There were recurrences in 6 patients, 4 of them being reoperated by us and healing in five to ten weeks without presenting any unusual problem. Table 1 summarizes the results of the different types of operations.

DISCUSSION

Our experience, as well as that of Buie[16, 17] and others,[18, 19] has shown that it is unnecessary to remove the sinus tract provided that all hair is removed and a wound is made that will heal without irregularities. Buie[16] postulated that after his marsupialization operation one of two methods of healing would take place — either the lining of the cyst, "which possesses practically the same histologic structure as that of true skin," would take part in the healing process (that is, become part of the surface epithelium) or, if the epithelium had been destroyed, the scar tissue that remained would be "just as efficient as any that may develop following excision of the entire cyst wall." More recently, Buie[17] stated that "the former possibility is rarely realized." We have never seen it occur. When the cyst lining has been left in place the wounds heal by filling in with granulation tissue, and after a few days are impossible to distinguish from those after total excision of the cyst except that the size of the defect is smaller. We believe that this diminution in the size of the defect is the chief advantage of leaving the cyst lining. Buie does not give the healing time in his cases. He states that the recurrence rate is negligible.

It seems incredible that the hair-producing epithelial lining of these cysts should be totally destroyed by infection in the great majority of cases. Grau's[20] work indicates that epithelium is not readily destroyed by infection. Yet if any area of such lining remains it should be the basis of a recurrence according to all established theories of the disease. We have previously stated our opinion that "recurrences following pilonidal sinus operations are not due to persistence of hair-producing epithelium but are true recurrences due to mechanical factors caused by collection of hair in an imperfect scar."[13] We have come to believe that most pilonidal sinuses develop in a similar manner and that there is practically never a hair-bearing epithelial lining. In many patients a congenital dimple of the skin in this area may be the point of origin. In the rest we can only surmise that some combination of stiff hair, tender or macerated skin, poor hygiene and possibly repeated trauma (as in the jeep driver) causes the initial penetration of the skin. Once an opening exists the loose hairs that collect in the sweaty gutter between the buttocks seem to find their way into it, as we have seen in the recurrent cases and as we repeatedly see in all open cases during the healing period.

We were led to this conviction by the three factors already mentioned: the long, midline, hair-filled recurrences, apparently originating in a depression in the scar, that we have seen in 9 cases after extensive excision and closure; the high percentage

of cures in our series and in Buie's when the cyst lining has not been removed; and the fact that the pattern of wound healing is exactly the same whether excision or incision has been performed. Once the belief in the congenital origin of these lesions is questioned other points against it come to mind.

Gross examination of the excised lesions shows that the epithelium is largely missing, and when present it is only at the site of the surface openings, as if it had grown down from the surface. Gage[7, 8] finds hair follicles and "immature hair shafts." Kooistra[6] describes hair in 52 per cent and hair follicles in only 9 per cent of excised lesions, but in our experience the hair is never attached to the follicles. It is usually a wisp of hairs, all about the same length, and the same color and consistence as the patient's body hair — not the fine, mouse-colored, crinkly, immature hair seen in a dermoid cyst. Stone[9] has described a case with hair that was "thin, fine, scanty in pigment somewhat like lanugo."

In figures previously cited on sacrococcygeal dimples in the newborn no difference in incidence was noted between the sexes. However, the ratio of males to females in patients with pilonidal disease coming for treatment varies from 3:1 to 7:1. We have seen no published statistics on symptomless pilonidal disease found on routine examination. Such figures have been supplied to us by Cowan,[21] of the Student Health Service of the University of Minnesota. In routine examination of 31,497 male students 365 were found to have pilonidal sinus whereas among 21,367 female students there were only 24 with pilonidal sinus. The average age of both groups was twenty-one years.

The fact that a relation exists between obesity and pilonidal sinus in males, although long our clinical impression, has been confirmed by analysis of figures from the University of Minnesota. It was found that 30,480 male students without pilonidal sinus averaged 103.12 per cent of "normal weight" (based on standard tables of normal weight for height and age) whereas 355 students with pilonidal sinus averaged 109.06 per cent of normal weight. This is a statistically significant difference ($P < 0.001$). Twenty-six per cent of the students without pilonidal sinus were 110 per cent or more of normal weight, and 46 per cent of those with pilonidal sinus were overweight (Fig. 3).

Although the 10:1 ratio of males to females is not a conclusive argument against the congenital origin of pilonidal sinus, it seems unlikely that such a difference would exist if the lesion were congenital. It also seems unlikely that obesity should correlate with a congenital lesion. On the other hand both factors fit the theory of mechanical origin.

Finally, reports of pilonidal sinuses found in other regions than the sacral have recently appeared in the literature. Smith[22] reported 4 "anterior pilonidal sinuses," 1 has been reported in the axilla,[23] and Patey and Scarff[24, 25] and Downing[26] called attention to pilonidal sinuses in barbers' hands. For none of these can a congenital origin be offered.

We cannot deny that some pilonidal sinuses may be of congenital origin. The ones connecting with the spinal canal undoubtedly are, but we believe that the theory of mechanical penetration of the skin by hair, with the formation of a foreign-body cyst, offers a more satisfactory explanation for the majority of cases. It is unlikely that further information can be gained from the study of embryologic specimens or of pilonidal sinuses excised surgically from adults. A study of sections of the tissues of the sacrococcygeal region taken at autopsy from children up to the age of twelve would reveal the true incidence of congenital lesions.

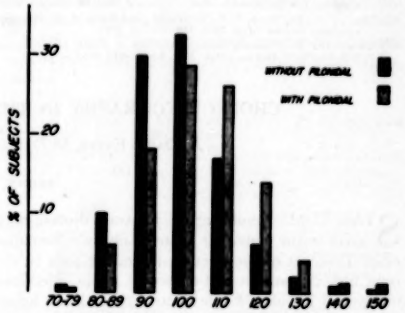

FIGURE 3. *Weight Distribution in 30,480 Male Students without Pilonidal Sinus Compared with That of 355 Male Students with Pilonidal Sinus (Based on Standard Tables of Normal Weight for Height and Age).*

SUMMARY AND CONCLUSIONS

Experience with different operations in 449 cases of pilonidal sinus is presented.

A simple method of primary closure for use in small, midline, uninflamed lesions is described. A method of treating all other cases by incision and packing is also reported.

The success of treatment without excision of the cyst lining, the finding of hair-filled recurrences after extensive excision, the pattern of healing after incision, the gross appearance of the hair in pilonidal sinuses, the ratio of males to females, the association of obesity and trauma and the finding of similar lesions in other regions than the sacral, all suggest that the majority of pilonidal sinuses are not congenital lesions.

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CHOLECYSTOGRAPHY IN THE PRESENCE OF LIVER DISEASE*

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SYMPTOMS simulating biliary-tract disease may arise in the course of acute or chronic liver disease. Diseases of the liver that produce pain in the right upper quadrant and otherwise mimic affections of the gall bladder or bile ducts include acute hepatitis, portal cirrhosis, acute or chronic passive congestion of the liver and hepatoma. Although the differential diagnosis between jaundice due to parenchymal liver damage and that due to biliary obstruction presents the problem in its most familiar and manifest form, the distinction in patients without jaundice is a problem frequently encountered in clinical medicine. Moreover, disease of the biliary tract may complicate hepatic disorders and vice versa. This study is limited to patients with liver disease in the absence of jaundice.

The use of the Graham-Cole test for visualization of the gall bladder is a standard procedure in the diagnosis of cholecystitis and cholelithiasis. Since it depends on the excretion of the dye by the parenchymal cells of the liver, it is essential to know whether, and to what degree, impairment of liver function interferes with gall-bladder visualization. Previous work by Bockus[1] indicated that a satisfactory gall-bladder shadow could be demonstrated by the cholecystographic technic in many nonicteric patients in spite of bromsulfalein retention of 25 per cent of the dye (the amount of dye given was 2 mg. per kilogram of body weight, and the thirty-minute test was employed). No statement concerning the number of cases or the regularity with which this degree of retention occurred was made. Blomström and Sondström,[2] also with the bromsulfalein test of 2 mg. per kilogram of body weight, found that retention of more than 40 per cent of the dye in the blood of nonjaundiced patients half an hour after its injection usually precludes the possibility of obtaining satisfactory cholecystograms. They also concluded that the absence of a gall-bladder shadow on cholecystography in spite of a bromsulfalein retention of less than 25 per cent shows that the obstruction is located outside the true liver parenchyma.

It was considered desirable to obtain more detailed information concerning the relation of hepatic damage, as revealed by the bromsulfalein test, to gall-bladder visualization as demonstrated by oral cholecystography. This information was considered especially necessary since the present standard test, which employs 5 mg. per kilogram of body weight over forty-five minutes, is more sensitive than that of 2 mg. over thirty minutes, which has been generally discarded. Moreover, the contrast preparations available for oral cholecystography have improved considerably since 1932 when Blomström and Sondström did their study.

After the intravenous administration of bromsulfalein in man, the dye has been recovered from bile within fifteen minutes.[3, 4] The maximal excretory rate of the dye is reached at forty-five minutes.[5] Tetraiodophenolphthalein and some of the newer water-soluble organic iodine compounds such as iodoalphionic acid (Priodax) appear in the bile and gall bladder in sufficient concentration after oral ingestion to permit the heavy halogen atoms to cast a shadow on the roentgenogram.

The extent of hepatic dysfunction as measured by bromsulfalein retention that interferes with adequate roentgenographic visualization of the gall bladder is the subject of this study.

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Method

Thirty-five nonjaundiced patients with abnormal bromsulfalein retention varying from 7 to 44 per cent were subjected to oral cholecystography. This group consisted chiefly of patients with compensated portal cirrhosis and hepatitis, established by clinical findings or biopsy or both; no history or symptoms of primary gall-bladder or biliary-tract disease could be elicited.

The standard 5 per cent solution was used. The test was performed with the patient under basal conditions; 5 mg. per kilogram of body weight was injected intravenously. A venous blood sample from the opposite arm was taken forty-five minutes later. The determination was performed with the Klett photometer, a green filter being used after the addition of 10 per cent sodium hydroxide to the serum.

Cholecystography was performed as the standard two-day test employing iodoalphionic acid. If there was failure of visualization after the first day's dose of 6 gm., the same amount of dye was given on the second day.

Results

The results are presented in Table 1. There were 23 patients with bromsulfalein retention of less than 20 per cent. There was normal visualization of the gall bladder in all these cases. The gall bladders of 5 patients with bromsulfalein retention varying between 20 and 23 per cent were faintly visualized.

The gall bladders of 7 patients with retention greater than 23 per cent were not visualized with iodoalphionic acid, even after a repeated dose of dye was administered on the second day.

No toxic effects resulted from the use of iodoalphionic acid in the presence of liver damage.

Discussion

Liver damage is often considered to be a contraindication to cholecystography. There is adequate evidence that this limitation of the use of the test is a relative and not an absolute one.[1, 2] Both the bromsulfalein test and oral cholecystography involve the excretory capacity of the liver. Although impairment of liver function affects both procedures, only the degree of bromsulfalein retention is of value as a test of liver function. Cholecystographic visualization has no clinical application in the estimation of derangement of hepatic function since it does not lend itself to quantitative determination, and more precise methods are available. However, it is important to consider not only the presence but also the degree of intrinsic liver disease when one is appraising the validity of the Graham-Cole test.

The present study indicates that visualization of the gall bladder by means of the cholecystographic technic is a practical procedure despite the presence of a moderate degree of parenchymal disease of the liver as measured by bromsulfalein retention. It is well known that varying degrees of hepatic enlargement may accompany cholecystic disease and that the occurrence of both hepatic and gall-bladder disease in the same person is not rare. In these situations, cholecystography and the performance of the bromsulfalein test may be essential in differential diagnosis.

The regular demonstration of adequate visualization of the gall bladder by means of cholecystography despite hepatic damage resulting in a bromsulfalein retention of less than 20 per cent has obvious clinical significance.

There were 2 cases in which progressive hepatic impairment was noted on successive admissions.

Table 1. *Findings on Cholecystography in 35 Patients with Hepatic Disease.*

Case No.	Diagnosis	Bromsulfalein Retention*	Cholecystography
		%	
1	Cardiac cirrhosis	12	Visualization
2	Lymphosa coma	32	Nonvisualization
3	Portal cirrhosis	30	Nonvisualization
4	Infectious hepatitis	28	Nonvisualization
5	Infectious hepatitis	17	Visualization
6	Portal cirrhosis	17	Visualization
7	Portal cirrhosis	44	Nonvisualization
8	Portal cirrhosis	20	Faint visualization
9	Cardiac cirrhosis	13	Visualization
10	Infectious hepatitis	7	Visualization
11	Chronic hepatitis	17	Visualization
12	Cardiac cirrhosis	20	Faint visualization
13	Portal cirrhosis	15	Visualization
14	Chronic hepatitis	28	Nonvisualization
15	Portal cirrhosis	7	Visualization
16	Carbon tetrachloride poisoning	8	Visualization
17	Cholangiolitic hepatitis	25	Nonvisualization
18	Cardiac cirrhosis	12	Visualization
19	Cardiac cirrhosis	12	Visualization
20	Cardiac cirrhosis	15	Visualization
21	Portal cirrhosis	38	Nonvisualization
	Probable hepatoma		
22	Infectious hepatitis	8	Visualization
23	Portal cirrhosis	17	Visualization
24	Portal cirrhosis	22	Faint visualization
25	Convalescence from infectious hepatitis	7	Visualization
26	Portal cirrhosis	12	Visualization
27	Portal cirrhosis	23	Faint visualization
28	Infectious hepatitis	12	Visualization
29	Portal cirrhosis	10	Visualization
30	Portal cirrhosis	11	Visualization
31	Lymphoma	10	Visualization
32	Chronic pancreatitis	17	Visualization
33	Portal cirrhosis	23	Faint visualization
34	Infectious mononucleosis	7	Visualization
35	Infectious mononucleosis	8	Visualization

*At the end of 45 min.

Normal visualization of the gall bladder was recorded when the bromsulfalein retention was under 20 per cent. The repetition of the cholecystogram on two subsequent admissions failed to visualize the gall bladder when the retention was 28 per cent in 1 case and 30 per cent in the other.

One patient had repeated admissions for Laënnec's cirrhosis with jaundice (serum bilirubin of 2.3 mg. per 100 cc.). The bromsulfalein retention in this patient was 7 per cent. Although the cholecystogram revealed normal visualization, numerous opaque calculi were seen. Perhaps the recurring jaundice was related to the passage of biliary calculi and

intermittent obstruction rather than hepatic decompensation.

Summary and Conclusions

Oral cholecystography in 35 patients with a bromsulfalein retention of 5 per cent or more gave satisfactory visualization of the gall bladder in 23 patients with retention of less than 20 per cent, and faint or no visualization in the remaining 12 patients with retention of 20 per cent or more.

The use of iodoalphionic acid for gall-bladder visualization in the presence of hepatic damage was without toxic effect.

Failure to visualize the gall bladder by the cholecystographic technic in patients with less than 20 per cent of bromsulfalein retention is suggestive of gall-bladder disease.

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MEDICAL PROGRESS

WAR WOUNDS (Concluded)*

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Care of Specific War Wounds

Arteries.[35] The successful primary repair of wounds of major peripheral arteries represents the most significant advance in the care of the wounded that has occurred in the Korean conflict. Some patients with wounds of major arteries in the body cavities do not survive long enough to receive treatment; however, this is not so in the periphery, where the surrounding tissues limit the amount of blood that is lost. In a recent series in Korea, Jahnke and Seeley[35] reported only 1 early anastomotic failure in the repair of 33 consecutive major peripheral arterial injuries. The amputation rate in this group was 8 per cent, in sharp contrast to a 45 to 50 per cent rate to be expected in similar arterial wounds treated by ligation. The ligation of a major artery is an admission of failure and should be done only as a last resort. A technical factor that deserves emphasis is that all the visibly damaged arterial tissue must be removed, in addition to 1 cm. of normal-appearing artery from each end of the vessel. This apparently normal arterial tissue on microscopical examination usually reveals evidence of intimal damage, rupture of the internal elastic membrane and herniation of portions of the media through the rents in the internal elastic membrane. It is plausible to assume that these microscopical changes might lead to thrombosis if the anastomosis were performed in this damaged tissue. Potts arterial clamps are used to occlude the vessels. The ends of the artery are then approximated and joined together with a No. 00000 silk suture placed as a continuous everting mattress stitch. If the arterial defect is too large to allow for repair by end-to-end anastomosis, the gap is bridged by an autogenous vein graft. The cephalic vein in the upper extremity and the greater saphenous vein in the lower extremity are satisfactory for this purpose. They are superior to the concomitant veins for the following reasons: they are approximately the same size as the artery whereas the concomitant veins are larger (the isodimensional graft provides for a laminar flow of blood with a minimum of turbulence and a decreased liability to thrombosis); they are thicker walled than the concomitant veins and are thus easier to work with; and their removal results in less interference with the venous return than the sacrifice of the concomitant veins. Because of the presence of valves in these veins, they must be reversed when placed in the arterial tree. The suture technic for the artery-vein anastomosis is the same as that for the artery-artery anastomosis. Dilatation of the fresh autogenous vein grafts in the periphery appears not to be a problem because of the heavy musculofascial support afforded these grafts by the surrounding and supporting tissues. Anticoagulant therapy and sympathetic blocks have not been used.

In cases in which amputation has subsequently been necessary despite an earlier attempt at arterial repair, the concomitant vein has usually been found to be thrombosed. The clinical course in these patients has most often been that of progressive swelling of the leg with engorged veins that will not collapse. This has persisted despite fasciotomy and has been aggravated by sympathectomy. This sequence of events recently led to the policy of repairing major concomitant veins at the time of initial surgery if they are damaged.

The exact incidence of "delayed" thrombosis to be expected after arterial repair is not known. At present, all that can be said is that thrombosis does occur, in the presence of a warm, viable limb. Such patients are being studied at the Walter Reed

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Army Medical Center, and many are having the obliterated segment removed and the defect bridged by use of a fresh autogenous vein graft. The thrombotic process, when present, usually extends from the anastomotic site both proximally and distally to the first major branch.

Thermal burns.[24, 26-28] Intensified studies of patients with thermal injuries since World War II have culminated in a clearer understanding of the underlying problems of management. Improvements in resuscitation, in methods of nutrition, in technics of skin grafting and in antibiotic therapy have brought about the survival of a fair number of patients with burns of over 40 per cent of body surface who would at one time have been doomed.

It is axiomatic that treatment of the deranged physiology takes precedence over the local care of the burn injury. The burn wound draws protein, electrolytes and water from the blood circulating through its depths and swells with edema. The deeper and more extensive the burn, the greater the fluid loss into the damaged tissues, and the more urgent the need to replace the depleted fluid resources of the body. The rate of edema formation is greatest in the initial hours after injury, reaching its peak somewhere between the thirty-sixth and the forty-second hour. After this time resorption begins and subsidence of the edema occurs. Fluid replacement consists of administration of a mixture of colloid and electrolyte solutions into the blood stream in such concentrations and volumes as to replace completely the water, electrolytes and protein lost into the wound. Water is given by mouth to maintain normal renal function. If therapy is prompt and accurate, disturbance of the normal extracellular-intracellular equilibrium is corrected and life goes on.

The extent of the surface area burned is a guide to planning the amount and kind of fluids needed. Renal output is the best guide by which to modify this plan. The greatest need for fluid replacement is in the first eight hours after injury, and this need diminishes with time; restraint is necessary during the resorptive phase, which occurs after about the forty-second hour. Too little fluid may result in anuria, and too much in pulmonary and generalized edema. A common error is to overestimate the extent of the burn and to be overzealous in fluid and electrolyte therapy. Experimental work indicates that the plasma loss at the sixth hour is approximately 1 cc. per kilogram per cent of body-surface burn. From these and other observations, certain estimates of fluid requirements can now be made.

The extent of the surface area involved by the burn can be estimated by a simplified modification of the Berkow scale, which divides the body into regions of 9 per cent or multiples thereof, as follows: head and neck, 9 per cent; each upper extremity, 9 per cent; anterior trunk, 18 per cent; posterior trunk, 18 per cent; each lower extremity, 18 per cent; and perineal area, 1 per cent.

On the basis of the extent of body surface involved, the following simple formula may be used as a guide to fluid-replacement therapy during the first twenty-four hours[29]:

Colloid	= 0.5 to 1.0 cc. per per cent body burn per kg.
Electrolytes	= 1 to 1.5 cc. per per cent body burn per kg.
5 per cent dextrose in water	= to maintain a urinary output of not less than 30 cc. per hour.

A clinical example (30 per cent body-surface burn in a 70-kg. man):

Whole blood or other colloid	= 0.5 × 30 × 70 =	1050 cc.
Electrolyte solutions	= 1.5 × 30 × 70 =	3150 cc.
5 per cent dextrose in water	= about	2000 cc.
		6200 cc.

Immediately after admission the injured patient's clothing is removed, the extent of the burn is mapped out according to the scale mentioned above, an estimate is made of the patient's weight, an indwelling polyethylene tube is inserted under local anesthesia into an accessible vein and a blood sample is obtained for hemoglobin and hematocrit determination as well as for typing and cross-matching, and, finally, an indwelling Foley type of catheter is placed in the bladder for hourly measurement of urine.

The type of colloid selected is determined largely by the extent and depth of the burn. In a patient with a 20 to 25 per cent body burn a plasma expander such as dextran appears to be as effective as plasma in the early treatment of shock. The risk of serum hepatitis attending the use of presently available pooled plasma has resulted in a tendency to substitute whole blood for plasma, in approximately two parts of whole blood to one part of plasma expander. Whole blood remains the backbone of intravenous therapy for the treatment of shock in the extensively burned patient.

The administration of electrolytes in the first days after injury currently tends to the administration of one third as Ringer lactate and two thirds as sodium chloride solution. Additional electrolytes may be given by mouth in the form of modified Haldane solution.

The major portion of the estimated first day's calculated requirements of colloid and electrolytes is given in the first twelve to eighteen hours after injury. Half these amounts is given for the second twenty-four hours. The adequacy of therapy is confirmed by the determination of urinary output, hemoglobin levels and the clinical response of the patient. In patients with respiratory injury or cardiorenal disease, or both, colloid and salt administration is limited to amounts that ensure a minimal urinary output of 30 cc. per hour. When the urinary output falls below this minimum, a test of the adequacy of fluid intake is the rapid intravenous

administration of 500 cc. of 5 per cent dextrose in water; if prompt diuresis follows, the fluid intake is c iou ly increas :d.

Local care of the burn wound is instituted when the condition of the patient is satisfactory. No anesthetic is employed. Pain is combated by the intravenous use of morphine or a morphine substitute in a dose of 8 to 10 mg. Unless it is cosmetically clean, the burn wound is gently cleansed with a bland white soap or detergent. Blisters are unroofed, débris removed, and detached epithelium cut away. No local applications of any sort are used. Systemic penicillin or other antibiotic therapy is given for about five days. Standard doses of tetanus antitoxin, tetanus toxoid, or both, are indicated. The prophylactic use of polyvalent gas-gangrene antitoxin is of doubtful value. The use of ACTH or cortisone is not recommended since recent observations indicate that fulminating spread of infection may attend their administration and, further, that their administration does not diminish the need of the burned patient for blood, colloid, electrolyte or water therapy.[38, 40]

The principal methods of local care of the burn wound at present include the use of absorptive compressive dressings and the exposure principle.[38] Pressure dressings are employed primarily for extensive and circumferential burns of the trunk and extremities, and the exposure method is indicated for burns of the head and neck, the perineum and other burns of such configuration (unilateral or flash burns) as to permit complete exposure of the injury without undue difficulty or discomfort. The coexistence of soft-tissue injuries or compound fractures and burns contraindicates exposure. In addition to the foregoing, minor burns that can be adequately dressed and burns in patients who must be transported over great distances are best managed by the dressing method. The universal burns dressing now tested in Korea and in various burns centers has been found to be practical, effective and time saving. This dressing, which comes in two sizes — 55 by 45 cm., and 55 by 90 cm., with two rolls of semielastic bandage in a sterile package — is satisfactory for emergency use, for it has adequate absorptive capacity, permits even application of compression and immobilizes the affected parts.

The nutritional requirements of burned patients are such that the patient's own appetite must not be relied on to provide adequate calories. In addition to the patient's oral intake, an emulsion having a high protein content is administered through an intranasal plast'c tube as a continuous intragastric gravity drip. As a rule such d ip feedings are instituted as soon after injury as the gastrointestinal tract will accept them.

The closure of the full-thickness burn wound by skin grafting must be initiated as soon as practicable. Resection of the eschar is feasible when the area involved is not extensive, and the risk of shock is not grea . Other technics of management of the eschar, such as enzymatic débridement, are now under investigation. The electrodermatome has simplified the rapid cutting of grafts of uniform depth. Storage in the icebox of extra quantities of skin in bottles containing 10 per cent human serum or in gauze moistened with saline solution permits skin dressing of small areas in which some of the original grafts have failed to take. The skin so stored remains viable for about three weeks.

Wounds of the chest.[41] Wounds of the thoracic cavity, before the use of body armor, comprised about 10 per cent of battle-casualty admissions to a hospital. Many factors have contributed to a lower mortality and morbidity from thoracic injuries in Korea: profit from the experiences of thoracic surgeons in World War II; rapid evacuation of the wounded from the battle area by helicopter and airplane; the proximity of the Army surgical hospital to the front lines; early resuscitation; the use of antibiotics; refinements in postoperative care; and the institution of physical therapy at the earliest possible moment so as to reduce functional disability to a minimum.

Immediate correction of physiologic disturbance of cardiorespiratory function is carried out at the battalion aid station according to established principles. Tracheotomy is performed when indicated. The sucking wound is closed immediately by sterile gauze compresses held in place by adhesive plaster. Pericardial effusion is treated by paracenteses and instillation of penicillin. Pressure pneumothorax and hemothorax, the most frequent complications of chest wounds, are treated preferably by repeated needle aspirations of the pleural cavity, and if need be by bronchoscopic clearing of blood and mucus from the tracheobronchial tree, by novocain injections of intercostal nerves and by blood transfusions. As much air or blood, or both, as possible is removed at the time of thoracentesis, and the procedure is repeated as often as indicated until the chest appears clear on physical and roentgenographic examination. After removal of fluid the antibiotic of choice is instilled, if it can be safely administered by this route. In the majority of cases the contents remain sterile, and the patient recovers completely without coagulation or organization of the fluid.

Decortication is the method of treatment for patients with significant clotted organized hemothoraxes or empyema, and, as in World War II, the operation is performed within three to five weeks of injury. It is noteworthy that 92 per cent of the patients who came to decortication in the Tokyo Army Hospital had previously had closed intercostal drainage tubes inserted in Korea that had been kept in situ for two to eight days. In many cases there had been ingress of infection about the tube or the tube was occluded by fibrin and clots and by pleural adhesions about the intrathoracic portion of the tube by the time the patient

reached the Tokyo Army Hospital. In this same installation only foreign bodies exceeding 1.5 cm. in greatest diameter were removed, unless, of course, the location was such as to be dangerous to the patient or to give rise to some pathologic change within the chest. Almost all foreign bodies in or adjacent to the heart, mediastinum or great vessels were removed. The removal of intrathoracic foreign bodies is accomplished by open thoracotomy through a posterolateral incision with resection of a rib. Whenever possible foreign bodies are left undisturbed for two or three weeks to permit subsidence of the local reaction, to facilitate location of the materials, and to minimize blood loss. By this time the general condition of the patient is such that the procedure is reasonably well tolerated.

Wounds of the abdomen.[4, 25, 29-31] Abdominal injuries in the main consist of penetration of the peritoneal cavity with single or multiple injuries of the contained viscera. Concomitant injuries, such as fractures and thoracic-cavity penetration, contribute materially to the mortality rate from hemorrhage and shock; irreparable destruction of vital organs, early peritonitis and associated injuries are often present. The initial decision is when to operate. If the state of shock does not respond rapidly to infusions of blood, it is fair to assume that there is continuing hemorrhage, peritonitis or other occult causes and that operation must be undertaken while resuscitative measures are being continued. The principles of operative management are the same as those established heretofore: the abdomen is explored through an adequate paramedian incision, with lateral retraction of the rectus muscle. Perforations of the stomach and small bowel are repaired by suture or resection and anastomosis, as the damage indicates; exteriorization and enterostomy are no longer practiced. Wounds of the large bowel are managed by mobilization of the perforated segment and exteriorization through a laterally placed separate incision. In the rectosigmoid area, where exteriorization is impossible, the perforation is inverted by suture and a proximal colostomy is performed. Colostomy is mandatory for perforation of the rectum; the wound is not sutured, and wide posterior drainage is established. Splenectomy is the operation of choice in injuries of the spleen. Wounds of the liver, if bleeding at the time of exploration, may be treated by suture or packing. Drainage is important, for there may be extensive seepage of bile from injured biliary radicles. A conservative approach is the rule for abdominal wounds involving the kidney. Nephrectomy is reserved for fragmentation wounds and continuing hemorrhage. Ureteral injury is managed by repair and catheter splinting, with diversion of the urine above the site of anastomosis whenever possible; otherwise, temporary exteriorization of the proximal end of the ureter and drainage of the retroperitoneal space are carried out. Perforation of the bladder is managed by repair, and diversion of the urine by suprapubic cystostomy and adequate drainage of the space of Retzius.

Wounds of the nervous system.[42] These wounds are handled in a similar manner to those in World War II. Brain injuries are considered a surgical emergency only if there is rapid hematoma formation not evacuated adequately through the wounds of penetration; in these cases emergency decompression is required. Otherwise, the emphasis is on definitive surgery at the time of initial operation. Thorough débridement is mandatory. Metallic foreign bodies are of concern only if they are large, are intraventricular, are moving, have carried in clothing and so forth, or have traversed sinuses. As mentioned elsewhere, brain wounds are firmly and absolutely closed, without drainage, after establishment of a dry field. Fascia lata is used for defects of the dura mater. No really satisfactory method of cranioplasty has yet been devised. Autogenous bone reabsorbs, and foreign bodies, such as tantalum, may work loose and lead to collections of fluid and sterile pus about them that will necessitate their removal. Brain abscess is now relatively uncommon, as is postoperative meningitis.

Wounds of the skeletal system.[30, 32, 34, 43] There have been some changes in the treatment of fractures since World War II. The method of dealing with comminuted fragments of bone is different. Formerly, only fragments attached to the periosteal shaft or with considerable musculature were conserved. Now, all fragments unless grossly contaminated are retained to act as chip grafts with the prospect that if adequate surgery is performed early under antibiotic protection, a great number of patients will be spared the complications of delayed union or nonunion and their sequelae. Another change in the treatment of fractures is the increasing use of intramedullary nails in selected cases of both simple and compound fractures of the femur. This is allowing easier transportability of the patients, more rapid recovery and earlier ambulation.[44, 45] However, the same complications attend the use of these agents in military surgery as in civilian surgery. Accordingly, discretion must be exercised in their employment.

Control of infection in compound fractures continues to be a problem, especially when the lower half of the leg is involved, arterial injury or expanding hematoma is present or the knee joint is involved or when débridement is delayed, inadequate or impossible.[46]

Advances in Prevention and Treatment of Complications of War Wounds

Shock. Certain advances have been achieved in the treatment of shock. These have largely consisted in the solution of the logistical problems pertaining to the supply of plasma, plasma expanders

and blood available even in the battalion aid station in quantities sufficient to allow for the rapid administration of volumes heretofore considered highly dangerous. However, despite the success of these massive transfusions in resuscitating some men from the so-called "irreversible" phase of shock, this therapeutic measure becomes empiric after the normal blood volume has been restored. What is desired is a normal *circulating* blood volume and not just a normal blood volume within the intravascular space. Little is actually known of the mechanisms controlling vasomotor tone. Until more is learned about the normal mechanisms, the present methods of overtransfusion must be continued for want of more specific therapeutic measures. Vasoconstrictors are often ineffective in this advanced stage of shock.

It is known that bacterial toxins may of themselves produce shock or render it refractory to blood-replacement therapy. Accordingly, the intravenous use of antibiotics in shock, especially oxytetracyline and chlortetracycline, deserves study in combating further liberation of bacterial toxins.

In the postoperative period, especially during the first three days, a syndrome, as yet without explanation, may develop. This usually, but not invariably, is seen in the severely wounded patient who has required large transfusions for initial resuscitation. This is a condition of "delayed" shock. It begins with a gradual fall in blood pressure and an increasing pulse rate, which continues until the patient's death twelve to twenty-four hours later in spite of every means of therapy. These have included massive whole-blood transfusions and the use of norepinephrine, the latter usually giving the patient a temporary elevation of blood pressure but soon becoming ineffective. Little is known of the mechanism of this manifestation of the body to massive trauma.

Post-traumatic renal insufficiency.[47] In the patient with post-traumatic renal insufficiency the biochemical changes of the uremic state develop much earlier than in the civilian patient with nephrosis. It may be that the patient with extensive wounds absorbs nitrogenous products from these damaged tissues. This condition is characterized by oliguria, proteinuria, persistent acidity of the urine, fixation of specific gravity of the urine at a low level, excretion of hemoglobin-like material, azotemia, hyperkalemia, hypertension and electrocardiographic changes. Early recognition of nephropathy can be made by the observation of four factors in every patient who is resuscitated from shock: the hourly and the daily volume of urinary output; the presence of benzidine-positive material in the urine; the level of nonprotein nitrogen, blood urea nitrogen or creatinine; and the systolic blood pressure. A symptom complex of oliguria, pigment excretion, azotemia and hypertension establishes the diagnosis. Because of the serious nature of this complication of severe shock and wounding, a special renal-insufficiency center has been established in an evacuation hospital in Korea for the study and management of patients who, despite a normal blood pressure, excrete a dilute urine in a volume of less than 500 cc. per twenty-four hours in the period after resuscitation. All patients with this complication are flown to the renal center, where they are carefully studied by experts in the field of renal physiology. Abdominal wounds, with or without multiple extremity wounds, carry a much greater threat of renal insufficiency than extremity wounds per se. In all severe injuries and when there is a doubt about the status of urinary excretion, a urethral catheter is inserted, and the hourly volume of urine noted, the minimum desired being 30 to 50 cc. As in thermal injuries, a test to distinguish between a prerenal oliguria caused by dehydration and a renal oliguria caused by kidney failure is provided by rapid infusion of 250 cc. of 5 per cent dextrose in distilled water intravenously and a chart of the hourly urine output. The former condition will respond with a diuresis whereas the latter will not.

The management of post-traumatic renal failure is designed to prevent edema on the one hand and hyperkalemia and tetany on the other. A precise system for intake-output recording is essential. Daily body weight is a useful check on the status of total body water content. Fluid administration must be restricted to prevent gain in weight. Intravenously given hypertonic (50 per cent) glucose in quantities sufficient to replace calculated as well as insensible losses of water furnishes the additional advantage of the protein-sparing effects of glucose. This may be administered by a polythene catheter inserted into the inferior vena cava via the femoral vein. When other measures fail, in severe states with high serum potassium levels and electrocardiographic evidence of hyperkalemia, hemodialysis with the "artificial kidney" is employed. This latter measure has proved life saving in many situations at the renal-insufficiency center in Korea. The function of the kidney is to remove toxic products, particularly excesses of extracellular potassium, which allow the patient to survive for longer periods than would be possible without the kidney. This additional survival time may provide the kidneys with a sufficiently long "convalescent" period to allow for their functional recovery. Many patients have required more than one dialysis. Associated problems of injury evolving around poor wound healing and decreased resistance to infection are now being intensively studied in patients who would previously have succumbed to their renal lesions.

Wound infections.[24, 48] War wounds characteristically are lesions in which tissue injury is extensive and contamination universal. Infection is thus an ever-present possibility, and it continues to be a threat until healing is complete. In wounds from

low-velocity missiles this inoculation is direct and is limited to the confines of the wound tract. However, in the large temporary cavity caused by high-velocity projectiles, because of the negative pressure in the cavity, it may be possible for contaminated material to be drawn into the wound cavity well away from the projectile's tract.[8] The degree of contamination is related to cavity size and characteristics of the wound of entrance and wound of exit.

Contamination of wounds in Korea with a wide variety of bacteria is universal at the time of the patient's arrival for definitive treatment.[37] Three general types of bacteria are found: the sporulating fecal bacteria, the nonsporulating fecal bacteria and the pyogenic cocci. Studies of Korean soil indicate that it universally contains numerous varieties of clostridia as well as other bacteria. Bacteriologic studies of the wounds show that other sources of contamination, in addition to soil, exist and are important. Although the types of bacteria found in the soil and in fresh wounds are practically the same as those found in previous wars and in civilian life[48-51] there appear to be several major differences:

In the first place, most clostridia found are of the proteolytic type, and the saccharolytic, systemically intoxicating types are less prevalent than those reported in studies in World War II and in civilian life. This is reflected in the relatively low incidence of classic gas gangrene. On the other hand, necrotizing infections typical of proteolytic clostridial infections are seen. When gas gangrene or progressive infection of muscle by mixed bacteria occurs, prompt resection of the devitalized tissues and vigorous supportive therapy are instituted.

Secondly, the hemolytic streptococci are generally of the nonpathogenic (fecal) variety. Cellulitis caused by hemolytic streptococci is uncommon.

Thirdly, pathogenic staphylococci are present in 22 per cent of the wounds.

Finally, coliform bacilli are found in about a third of the cases; *Proteus Vulgaris* (*Bacillus proteus*) and *Pseudomonas aeruginosa* are common.

Active proliferation of these bacteria may be demonstrable in muscle planes away from the wound tract as early as six hours after injury. In about 10 per cent of cases of severe injury with shock, there is a transient bacteremia.

Wound infections are largely preventable by the employment of proper débridement at initial surgery and by the administration of antibiotics during the first five to seven days after injury. So far as tetanus is concerned, the success of a specific immunization program is demonstrated by the almost complete absence of this complication in wounded men. The prevention of infection in extremity wounds is made difficult by the following conditions: delay in, omission of or inadequacy of initial wound surgery from whatever cause; arterial insufficiency, venous obstruction or lymphedema; widespread tissue destruction such as that produced by a high-velocity missile; and oliguria and anuria complicating resuscitation in the patient with multiple injuries.

In penetrating wounds of the abdomen, infection is still a frequent and serious complication. This is particularly true when peritonitis is already established at the time of initial surgery, gross contamination of a retroperitoneal hematoma occurs, and penetrating abdominal injury is complicated by anuria. Because of the insidiousness of postoperative infections and the relative lack of local signs, close observation of the patient is necessary if peritonitis is to be diagnosed before the condition is far advanced.

In Korea the main reliance for antibiotic prophylaxis and therapy had been on procaine penicillin in dosage of 300,000 to 600,000 units, with or without 0.5 gm. of streptomycin administered intramuscularly. A recent appraisal of this regimen indicated that it was inadequate in several respects. These are as follows:

The absorption of intramuscularly administered antibiotics is retarded and erratic in shock.

The blood and tissue levels obtained are significantly lower both by their rate of administration and with this type of penicillin than with aqueous penicillin given intravenously. Studies of the concentration of penicillin in wounded tissues showed an average level of only 0.23 units per gram.[37] In the presence of tissue necrosis the concentration was even lower. In some cases no penicillin was found.

The lower the concentration of penicillin in the tissues, the greater the number and variety of mixed bacterial species present.

Culture sensitivity studies have revealed that a high percentage of the clostridia isolated from wounds in Korea have not been inhibited by this average concentration of 0.23 units of penicillin per gram of tissue.

This therapeutic regimen did not prevent colonization of the wound by bacteria in the presence of incomplete débridement.

The data currently available support the impression that the combination of penicillin and streptomycin as presently employed is successful in inhibiting infection by highly sensitive bacteria, such as the hemolytic streptococcus, but is inadequate against infections caused by mixtures of other less sensitive or resistant organisms. The course of peritonitis or proteolytic infections of muscle has not appeared to be influenced by this type of antibiotic treatment as by more intensive therapy.

Because of these above findings, the currently employed antibiotics regimen has been changed as follows:

For patients requiring intravenous administration of fluids (those in some degree of shock), aqueous penicillin G should be given with the fluids at the level of the battalion aid station as soon after wounding as possible in doses of 500,000 to 1,000,000 units every eight to twelve hours, depending on the severity and multiplicity of the injuries. Patients not in shock are given intramuscularly 600,000 units of procaine penicillin with 200,000 units of aqueous penicillin G added, and the injections are repeated at intervals of eight to twelve hours.

Streptomycin in doses of 0.5 gm. should be given concomitantly by vein.

After twenty-four hours, if the blood pressure is no longer reduced, the same agents should be given intramuscularly.

After seventy-two hours, the antibiotic therapy should be revaluated and changed as indicated. Emergence of drug-resistant forms may occur in war wounds as in other types of wound suppuration.

Streptomycin therapy should be discontinued after five days.

If antibiotic treatment is indicated after five days, one of the broad-spectrum drugs should be considered.

In penetrating wounds of the abdomen and gross peritoneal soilage, as in patients with extensive devitalization of muscle, oxytetracycline, chlortetracycline or chloramphenicol should be given in doses of 0.5 gm. intravenously at the time of the initial surgery along with penicillin in the doses mentioned above and continued as indicated. When the blood pressure is normal and oral intake is possible, the broad-spectrum agents should be given by mouth in doses of 0.5 gm. every four to six hours. This therapy should be reviewed every seventy-two hours and varied or discontinued as indicated.

Arteriovenous fistulas and aneurysms.[52, 53] The incidence of arteriovenous fistulas and aneurysms has decreased sharply with the policy of repairing the damaged artery at the time of initial surgery in the forward areas. All Army personnel with arteriovenous fistulas and aneurysms are sent to the Walter Reed Army Medical Center for treatment. Since the beginning of the Korean conflict over 180 patients have been treated. Of the last two thirds of the fistulas and aneurysms involving major vessels, all have been repaired by primary anastomosis or vein graft. The functional results obtained with restoration of arterial continuity after excision of the fistulas are far superior to those obtained with quadruple ligation as the method of treatment. The same statement may be made for the restoration of arterial continuity, as opposed to ligation of the artery, as the method of treatment of aneurysms. The aneurysmal sacs are left in place but dependent drainage is established. The anastomotic technic is similar to that employed for the repair of major peripheral arteries, as mentioned above.

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MEDICAL INTELLIGENCE

NEUROFIBROMA OF THE DIAPHRAGM

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PRIMARY tumors of the diaphragm are rare, less than 40 cases having been reported in the literature. In 1949 Clagett and Johnson[1] analyzed reports of 30 cases of diaphragmatic tumor and, when information was available, described the method of diagnosis. Sixteen of these cases were discovered at autopsy; 12 were diagnosed during life so that partial or complete surgical removal could be carried out, and no mention was made of the method of diagnosis in 2. In 1950 Samson and Childress[2] reported 5 additional cases, making 35 in all, of which 17 were benign and 18 malignant.

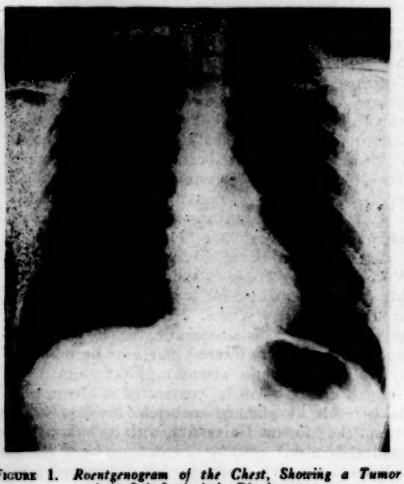

FIGURE 1. *Roentgenogram of the Chest, Showing a Tumor of the Left Leaf of the Diaphragm.*

With the increasing use of x-ray films in the diagnosis and study of pulmonary disorders and the ever-expanding use of routine chest survey films, tumors of the diaphragm should be detected before autopsy more frequently in the future. The case reported below is an example of a small, benign tumor of the diaphragm discovered incidentally on a chest film taken because of suspicion of pneumonitis. It is considered to be of interest because of the rarity of the tumor and also as an example of a condition that may become more frequently discovered with the increasing routine use of chest films.

CASE REPORT

E. S., a 26-year-old woman, was admitted to the hospital on May 11, 1953, because of a small shadow in the left-lower-lung field. Her general health had always been good. In the previous February she had had an upper respiratory infection with a moderate fever. She lived in the country, and it was difficult for her doctor to follow her closely at home so that she was taken to the local hospital. Her symptoms subsided within a few days, but an x-ray film of the chest before discharge showed an area of density, 2 x 1 cm., near the dome of the left leaf of the diaphragm (Fig. 1). Studies for tubercle bacilli in the sputum were negative. The patient remained asymptomatic, but a 2d film of the chest 2 months later revealed no change in the size or shape of the shadow. The patient was then referred to the Massachusetts General Hospital for treatment.

FIGURE 2. *Gross Specimen after Removal. The tumor has been cut in two, showing both cut surfaces.*

Physical examination disclosed a slender but well developed woman with no apparent abnormalities. Laboratory studies were normal except for a hemoglobin of 11.8 gm. per 100 cc. An x-ray film of the chest demonstrated a smooth-margined area of density, 2 x 1 cm., extending from the middle portion of the dome of the left leaf of the diaphragm. It moved synchronously with the diaphragm and was considered extrapleural in location. It was regarded as probably a primary tumor of the diaphragm of a benign variety, but a small hernia could not be ruled out. The lungs were otherwise clear.

After preparation with a transfusion, an exploratory thoracotomy was performed, with excision of a small, firm tumor of the diaphragm (Fig. 2.) This proved to be a neurofibroma on

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microscopical examination (Fig. 3). The diaphragm was repaired with interrupted sutures.

The patient made an uneventful and satisfactory convalescence and was discharged on the 12th postoperative day.

Discussion

Of the tumors of the diaphragm described in the literature, we have been able to discover only one other report of a neurofibroma.[2] Since nearly half

Figure 3. *Microscopical Section.*

the tumors of the diaphragm are malignant, it seems wise to emphasize the need of early diagnosis. Surgical resection offers no great technical difficulties if attempted before the tumor becomes too large and is essential for both diagnosis and cure.

Summary

A case in which the diagnosis of neurofibroma of the diaphragm was made during life is reported. The necessity of early diagnosis and surgical resection is emphasized.

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DOCTORS AFIELD

Anton Chekhov

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ONCE, in his later years, Anton Chekhov was walking with a friend after a rainstorm. "Someone ought to write a vaudeville sketch," he said. "Two people wait in an empty barn for the rain to pass. They joke, they laugh, they dry their umbrellas, make love to each other. Then the rain stops, there is sunshine, and suddenly he dies from heart failure."

"Good heavens!" exclaimed his companion. "What kind of vaudeville would that be?"

"Well," he answered, "such is life, is it not? We joke and laugh and then — bang! All is over."[1] Chekhov's own life was like this — active and amusing, but all too brief.

Born in 1860 into the large family of a poor shopkeeper, he grew up in the village of Taganrog, in the south of Russia. Though isolated and dull, Taganrog had a passable library and an excellent theater. Anton frequented both, even though school children were forbidden to attend theatrical performances. He eluded the villainous inspector with a liberal use of false beards and dark spectacles. His schoolmates sought out Anton for his skill in telling stories and impersonating pompous adults.

His father, whose business failed when Anton was sixteen, had to leave town hidden in the bottom of a cart to avoid being arrested by his creditors. Within a year the rest of the family except Anton followed; he remained in Taganrog for three years to finish secondary school. At some point between his sixteenth and his nineteenth year, he decided to study medicine. His interest may have been kindled by the sympathetic attention given him by a German doctor when he contracted a severe chill. As a result he planned to rejoin his family and attend the Moscow University, with its outstanding medical faculty.

Even his family's despairing letters were not preparation enough for his shock on arriving at their quarters. They were established in a damp basement in Moscow's red-light district. The father had been out of work for long periods and now earned a pittance as a wholesale clerk. Anton, best fitted by temperament for responsibility, took up the task of supporting 6 or 7 people in addition to beginning his five-year medical course. He found that he could earn a few rubles a week by writing stories for the humorous magazines.

One day in 1882 the family finances took a definite turn for the better. Anton was walking through the streets when he heard a shout, and a sledge drew up

beside him. Nicholas Leykin, editor of the Petersburg magazine *Fragments*, drew him to a tavern and asked him to write for his magazine. Not only were his terms advantageous but also he differed from most other editors by settling his accounts promptly. This arrangement served to increase Chekhov's literary output and to improve its content. Leykin's editorial policy was clearcut: "Keep it short and funny." Anton began to develop the economy of expression that later characterized his best work.

At the age of twenty-four, he graduated from medical school. He went to Voskresensk that summer, where his brother Ivan taught school, and

Anton Chekov (Reproduced from Toumanova[1] with the Permission of the Publishers).

gained experience by assisting the head of a nearby hospital and by taking over a doctor's practice in another town for a month or two. His writing now claimed more of his attention, and the resulting product was of greater literary merit than the frivolous stories of his first years. His work received comment in literary magazines, and he formed a very close friendship with Suvorin, editor of *New Time*. Ahead of him two fields of endeavor lay open, but he made no forthright decision between them.

He notified Leykin that his practice was "turning up little by little." Later he wrote to Suvorin: "You advise me not to hunt two hares at once, and not even to think about my medical profession. Why not? I feel more cheerful and satisfied when I realize that I have two things to do instead of one."[3]

The literary critics were unkind, for the most part, but his reputation continued to grow, and in spite of them, he was awarded the Pushkin Prize for Literature by the Academy of Sciences. At yearly intervals collections of his stories appeared and sold moderately well.

It was almost inevitable that Chekhov's medical practice should decline upon such widespread recognition of his literary skill. In a way this was fortunate, because his health showed signs of serious deterioration. With all his medical background, he ignored the warning of hemoptysis and overlooked the significance of his consumptive symptoms during the next thirteen years.

In the summer of 1889, his brother Nicholas died of tuberculosis. Anton had been very close to him, and the blow shook him badly. At last he found diversion in planning his great trip across Siberia to Sakhalin.

The penal system of Tsarist Russia included colonies of convicts in Siberia and the more recently instituted one on Sakhalin Island, which lies in the north Pacific. The journey, made in the days before the Trans-Siberian Railway, promised to be very arduous. His motives for undertaking it were complex, but he felt a compulsion to "repay a debt to medicine" by this humanitarian study of penal conditions. He read everything he could lay hands on that pertained to Sakhalin, packed his trunk, accepted a bottle of cognac from his friends to be drunk on the shore of the Pacific Ocean, and set off in April, 1890.

It was a succession of carriage stages and crossings by ferry. Twice he came close to perishing, but at last he had enjoyed his cognac. Chekhov was well received on the island and at once began his labors. Given free access to all except the political prisoners, he set about making a personal census of the entire population. By visiting each convict and filling out a small card, he had the opportunity of meeting as wide a range of people as possible. Eventually, he collected about ten thousand cards. He was depressed by the unspeakable brutality, filth and hunger prevalent on the island, and he found repeatedly that no attention whatever was given to the moral rehabilitation of the prisoners. He left by steamer in October, calling at Hong Kong, fighting a storm in the China Sea, and returning home by way of Ceylon, Suez and Constantinople.

Chekhov was pleased with his adventure, and, after a brief tour of Western Europe with Suvorin, he sat down to present his findings in a scientific paper. This sociologic study became the basis for eventual reform. He was soon occupied with collecting books for the children of Sakhalin and with famine relief in Russia itself.

Troubled by his persistent cough, by incessant visitors and by city living in general, he retired to a farm in the country to the south of Moscow. His neighbors hastened to consult him for their aches

and pains, since doctors were very scarce in the provinces. He corresponded with that giant of Russian letters, Leo Tolstoy, and went to see him. The two did not agree on philosophical questions, but they met again repeatedly and remained friends.

The short stories marked a new departure in Russian literature. Chekhov's style consisted of a succession of significant details, recorded with the detachment and "impassivity of a physician." His writing covers every level of Tsarist society, among the best known stories being "Ward No. 6," "In the Ravine" and "The Steppe." He was never a partisan in any cause, and yet his work as a whole shows a strong condemnation of existing conditions and an underlying hope of improvement. It is an accurate and complete survey of the social order that preceded the Revolution. The stories influenced such modern writers as Sherwood Anderson, Bernard Shaw and Ernest Hemingway.

His love of the theater played a large part in Chekhov's last years. He joined with the Moscow Art Theater in a movement to bring new life to the stage. Accordingly, his major plays, *The Seagull*, *The Three Sisters* and *The Cherry Orchard*, relied on valid character portrayal rather than on the older devices of wild dramatic action. Like his stories, they tended to be sophisticated social commentaries.

Anton built a villa at Yalta for the sake of his health. During rehearsals of the Moscow Art company, he fell in love with one of the leading actresses, Olga Knipper. They were married in 1901, but their time together was unfortunately limited. He could leave Yalta for Moscow only in the summer, and they agreed that Olga should not give up her career. Anton's health declined further, even with the care he finally gave it, and he died at a German health resort at the age of forty-four. Olga was left childless.

The final touch was provided by an oversight in arrangements for transporting his body back to Russia. It traveled in a railway car marked "Fresh Oysters." How Chekhov would have enjoyed the irony of that incident!

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Massachusetts Physicians Art Society

20. Gloucester Sloops

Exhibited at American Medical Association San Francisco, 1950

Exhibited at Annual Meeting of the Massachusetts Medical Society, Boston, 1952

Free Brush and Ink by
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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

BENJAMIN CASTLEMAN, M.D., *Editor*

VIRGINIA W. TOWNE, *Assistant Editor*

CASE 39491

PRESENTATION OF CASE

First admission. A sixty-three-year-old housekeeper was referred to the hospital because of the sudden onset of aphasia.

About two months before admission the patient had vomited once while asleep, but otherwise she had been well until two days before admission, when incoherence of speech suddenly developed. Her friends observed that she frequently misused words, and that she had great difficulty in understanding what was said to her. She gave no evidence of headache or other discomfort, and there was no impairment of the use of the limbs. This condition remained unchanged over the following day, and she was referred to the hospital.

Physical examination revealed a well developed and well nourished woman who often misused words but did not seem aware of her mistakes. She could not follow simple verbal instructions, spoken or written, but could carry out a movement when shown how to perform it. The heart was not enlarged and there were no murmurs, but the cardiac rhythm was totally irregular. The pupils were unequal (right 4 mm., and left 3 mm.), but reacted promptly to light and in convergence. The right nasolabial fold was slightly flattened. The cranial nerves were otherwise intact. There was no motor impairment of the limbs or trunk and no reflex or sensory changes. The remainder of the physical examination was negative.

The temperature was 100.2°F., and the respirations were normal. The radial pulse was 100, and the apical heart rate 116. The blood pressure was 130 systolic, 50 diastolic.

The routine urine and blood determinations were normal. The serum nonprotein nitrogen was 23 mg. per 100 cc. A blood Hinton test was negative. Lumbar puncture gave clear, colorless spinal fluid under a pressure equivalent to 80 mm. of water. The fluid contained 9 lymphocytes and 30 crenated red cells per cubic millimeter. The total protein was 99 mg. per 100 cc., and the gold-sol curve 0011242110; the Hinton test was negative. A roentgenogram of the chest showed the heart to be normal for the patient's age. The aorta was somewhat tortuous. Both leaves of the diaphragm were low and flattened. The right middle lobe was decreased in size and contained what was interpreted after fluoroscopy as a small area of atelectasis. There was also an irregular linear scar adjacent to the left border of the heart. Skull films were normal. An electroencephalogram on the seventh hospital day gave a markedly abnormal record, with high-voltage, slow waves on the left, particularly anteriorly and temporally.

Digitoxin was given and on the third hospital day the cardiac rhythm reverted to normal at a rate of 80 per minute. An electrocardiogram at this time showed normal rhythm, and was interpreted as indicating digitalis effect.

The aphasia steadily improved from the time of admission. There were two brief episodes of slight headache with vomiting. A second lumbar puncture ten days after the first gave clear spinal fluid under low pressure that contained about 10 lymphocytes per cubic millimeter; the total protein was 128 mg. per 100 cc., and the gold-sol curve was similar to the first. On another lumbar puncture a week later the spinal fluid was under a pressure equivalent to 160 mm. of water and contained no cells; the total protein was 50 mg. per 100 cc., and the gold-sol curve was normal. The patient was discharged on the twenty-seventh hospital day to continue convalescence at home.

Second admission (eighteen months later). Her use and comprehension of language continued to improve. On a visit to an ophthalmologist because of some persistent difficulty in reading, she was found to have a right homonymous hemianopia. An electrocardiogram about nine months after discharge again showed auricular fibrillation at a rate of 110 per minute. Four months before the second admission, the patient observed that her "mouth was pulled to the right" for about two weeks, after which it reverted to normal. Two months later this recurred, without any additional new symptoms. On examination at this time in the Out-Patient Department, she was found to have a weakness of the left lower side of the face. The other physical findings were unchanged; auricular fibrillation was still present. Eighteen months after the first admission, she was readmitted to the hospital with the complaints that for about two weeks she had been vomiting about four times a day and that for a week her mouth had been "pulled to the left." More recently, she had had a sensation of "whirling around," which was not more precisely described.

On examination she was correctly oriented, but gave a rambling, disconnected account of her symptoms, and frequently mispronounced or misused words. There was well sustained nystagmus

on right lateral gaze. A right homonymous hemianopia was present as before. The optic fundi were not remarkable, and the pupils were normal. The right corneal reflex was absent, and there was indefinite impairment of sensation over the right upper part of the face. Jaw movements were normal. There was a fairly complete paralysis of the right side of the face and loss of taste on the right anterior two thirds of the tongue. Hearing was decreased in the right ear. The palatal arch was lower on the right than the left, but phonation and swallowing were normal. The tongue was protruded in the midline. Gait was generally unsteady, with a tendency to fall backward. There was slight weakness of the right hand and of dorsiflexion of the right foot, and the heel-to-knee-to-shin test was clumsily performed on the right. Tendon reflexes were slightly brisker on the right side than the left, except that the right ankle jerk was absent. The plantar reflexes were flexor or absent. There were no sensory changes in the trunk or limbs. The lungs were clear. Auricular fibrillation persisted. There was a high-pitched Grade 2 systolic murmur at the apex. The remainder of the physical examination was unremarkable.

The temperature, pulse, respirations and blood pressure were normal.

The spinal fluid, which was under a pressure equivalent to 110 mm. of water, was clear and contained 20 lymphocytes per cubic millimeter and 76 mg. per 100 cc. of protein; the gold-sol curve and Hinton test were negative. X-ray films of the chest were unchanged from the previous examination. A gastrointestinal series was negative. An electrocardiogram demonstrated auricular fibrillation and digitalis effect. An electroencephalogram again disclosed a slow-wave focus in the left frontotemporal region; the abnormality was considered to be not as marked as during the previous admission.

On the eighth hospital day a hearing test showed absolute deafness in the right ear. Stimulation of the labyrinths with cold water revealed no reaction on either side. The vocal cords were seen to move normally. Dicumarol was given, and the prothrombin time was maintained at about 15 per cent. On the thirteenth hospital day, in addition to previous physical findings, nystagmus on lateral gaze to either side and on upward gaze was found. Sensation was thought to be diminished on the right side of the pharynx. The patient fell to the right on walking. She continued to vomit guaiac-negative material several times daily. Auricular fibrillation persisted despite digitalis and quinidine therapy. X-ray films of the skull on the fifteenth hospital day were normal except that the dorsum sellae appeared somewhat porotic. The structures at the base appeared normal. On the twenty-ninth hospital day the tendon reflexes were more active in the left arm than in the right; knee jerks were equal; ankle jerks were absent. The left plantar reflex was now equivocal, and the right normal. Neurologic findings were otherwise unchanged except that the sensory loss on the right side of the face was more pronounced than at the time of admission. On the thirty-second hospital day, an operation was performed.

Differential Diagnosis

Dr. William H. Sweet*: The right homonymous field defect was mentioned for the first time on an examination during the interval between admissions. Did she have a normal field at the first admission?

Dr. Edward P. Richardson, Jr.: It was checked off as normal in the record.

Dr. Sweet: That field defect is, as far as I can see, the only evidence of advance of the lesion described in the first admission.

This first episode of abrupt onset of aphasia accompanied by slight weakness of the right side of the face in a right-handed patient — I assume that she was right-handed — at this age in the presence of auricular fibrillation suggests a vascular episode involving presumably the branches of the left middle cerebral artery. This ictus may have been due to embolism from thrombosis in the cardiac atrium, to a thrombosis arising *de novo* in the left middle cerebral artery or one of its branches, or somewhat less likely to a thrombosis in the main internal carotid artery. In the last few years since we have been doing more arteriograms we have become aware that the syndrome of occlusion of the internal carotid artery is more protean in its manifestations than we had suspected; that the symptoms may be confined to one branch of that artery even when the main trunk is involved; and that if the symptoms are confined to one branch, they are more likely to be confined to the middle cerebral rather than the anterior cerebral branch because of the greater number of anastomoses between the two anterior cerebral arteries. The findings on lumbar puncture — 10 lymphocytes per cubic millimeter in the spinal fluid — are quite characteristic of an episode of infarction in a cerebral hemisphere that is not large. The only finding on examination at that time that makes me worry about some other diagnosis is the elevation of the total protein in the spinal fluid, especially when it went up to 128 mg. per 100 cc.; however, it returned to a normal level by the time of discharge from that admission. The only evidence of worsening of the lesion in the left cerebral hemisphere after discharge was the right homonymous field defect; this leads me to think that she then had a vascular lesion and not a tumor. The long duration of the gradual subsidence of these symptoms also tends to exclude a tumor in the left cerebral hemisphere at this time.

May I see the x-ray films now?

*Associate visiting neurosurgeon, Massachusetts General Hospital.

DR. JOSEPH HANELIN: The chest examinations done nineteen months apart show no essential change in that period. There is atelectasis of the right middle lobe and a small linear scar along the left margin of the heart.

DR. SWEET: Would you hazard a guess about the cause of that scar?

DR. HANELIN: This presumably could be due to an old infarct. The last films demonstrate exactly the same findings; the heart seems larger because this is an anteroposterior rather than the conventional posteroanterior view. The plain films of the abdomen show normal visceral outlines. The upper gastrointestinal series reveal no significant abnormality. Two sets of skull films, also nineteen months apart, disclose approximately the same findings, and any positive findings that we see are certainly minimal. There is a question of a slight pineal shift to the right side, but the pineal gland remained in the same location in the two examinations. Also, any erosion or decalcification of the dorsum sellae is minimal and questionable. There is no abnormal intracranial calcification.

DR. SWEET: The next two neurologic episodes of which the patient was aware occurred four and two months before the second admission, during which she had some transitory weakness of the left side of the face. This symptom bears out the thought that she was probably having multiple vascular lesions in the right cerebral hemisphere, perhaps even smaller ones than previously. Then she began to warn us by presenting an entirely different type of symptomatology immediately before and during the second hospital admission. She apparently also had some recrudescence of the aphasia — she was mispronouncing and misusing words. This may have been the phenomenon that we see in patients who have a disease of the cerebrum that leaves a scar. Then when the patient has some other disorder that weakens him — it need not even be in the central nervous system — the old signs may again become manifest. I have seen this happen even when the patient had an attack of influenza without any evidence of a new vascular accident in the brain. I do not know, therefore, that I can decide that the patient was having further evidence of progression of the left cerebral lesion from the fact that the aphasia seems to have been troubling her somewhat more.

She had, in addition, signs of involvement of the cerebellopontine angle. The absent corneal reflex, although seen occasionally in cerebral lesions involving the sensory cortex, is much more typical when the trigeminal nerve itself is affected somewhere in its course. The same statement applies to the diminished sensation over the right upper part of the face; the progression of the sensory loss confined to the face noted during the hospital course also indicates that the trigeminal nerve rather than a cerebral sensory pathway was affected. This conclusion is supported by the fact that she had a complete paralysis of the right side of the face, indicating that the facial nerve was affected somewhere between its source of origin in the brain stem and the point of departure of the chorda tympani in the bony canal for the nerve. The loss of taste in the right anterior two thirds of the tongue (seventh nerve), the deafness in the right ear (eighth nerve), the diminished sensation of the right side of the pharynx (ninth nerve), the weakness of the right levator veli palatini (tenth nerve) and the tendency to deviate to the right and to fall to the right in walking that occurred later on, all point to a lesion in the right cerebellopontine region. I do not know whether the weakness of the right hand and the weakness of dorsiflexion of the right foot represent a sign that had occurred on the basis on which I have explained the recrudescence of the aphasia, on the basis of involvement of the left pyramidal pathways to the brain stem or on the basis of some disturbance of cerebellar pathways with which this degree of weakness might be consistent. I do not think the right hemiparesis will help me particularly in deciding what was going on here. The clumsiness in the carrying out of the heel-to-knee test is further evidence of a lesion in the right cerebellopontine angle. I do not know why the right ankle jerk was absent and then, later, the left ankle jerk became absent. This suggests the possibility that a lesion in the posterior fossa seeded itself down into the cauda equina, if it was a neoplasm, or, since this woman had multiple vascular episodes, that the blood supply to the nerve pathways responsible for the ankle jerks had been involved. Since the spinal-fluid protein increased while the electroencephalographic changes seen on the first admission improved rather than worsened and the right lower cranial nerves became severely afflicted, I am more inclined to think that she had not had another vascular episode in the left cerebellar hemisphere, and to concentrate my attention on the brain stem and cerebellopontine angle.

To help me in determining whether or not the lesion was intrinsic or extrinsic to the brain stem I have a number of other findings. The fact that the caloric response was absent on both sides is not conclusive evidence of an intrinsic lesion of the brain stem, because the cranial nerves are quite vulnerable to compressive lesions in the cerebellopontine angle on the opposite side; indeed, cases have been reported in which all the cranial nerves were affected on the side opposite the neoplasm. The fact that the nystagmus had become more extensive and included nystagmus on upward gaze as well as on lateral gaze to either side suggests that the lesion was in the brain stem; vertical nystagmus occurs somewhat more frequently with a lesion in that site than with one in the cerebellopontine angle, according to our own authority on the subject, Dr. David Cogan.[1] I am assuming that

the direction of motion on upward gaze here was in the vertical plane although this is not specifically stated. The vomiting that she had intermittently throughout the whole illness was perhaps related to irritation of the vagal nuclei. Late in the course in the hospital, before operation, signs of involvement of the right pyramidal tract were beginning to develop. The tendon reflexes for the first time became more active in the left arm than in the right, and the left plantar reflex became equivocal whereas they had both been normal before.

I have to consider the disorders that may cause first of all an extrinsic lesion in the cerebellopontine angle. In a patient who had already had a number of vascular episodes, one might suspect that this would be on the basis of an aneurysm. We know that the tortuous, S-shaped, basilar aneurysms can become so large as to spread out into the angle and can give signs of this type; the spherical basilar and vertebral aneurysms can do the same thing. Although this portion of the history relating to the lesion in this region was not recurrent and had no episodic character, many of these aneurysms tend to grow as a tumor does and do not give episodic manifestations, such as those recently described by Denny-Brown and Foley.[3] The principal neoplasms in this region are rather slowly growing; in only 2 of 20 cases with meningiomas in the angle of the lateral recess that Cushing[2] reported did the patients have symptoms relating to that region that were less than one year in duration, and yet this patient's symptoms were only a few weeks in duration. The same slow growth applies to an ever greater degree to acoustic neuroma although I have seen a patient with an acoustic neuroma who had symptoms that were less than one month in duration. The epidermoid or pearly tumor tends to have a much longer course so I think I can definitely exclude that. Boldrey[4] recently described a very encouraging kind of lesion in this region — simply an accumulation of cystic fluid; he reported on 5 cases in the last five years in which he explored the angle expecting to find a sizable tumor and found that all he had to do was release some watery, clear fluid and the patient's substantial signs and symptoms cleared up. In 1 of these 5 patients the duration of the symptoms was very brief. Such cases as that lead one to look at the cerebellopontine angle in patients of this type even though one is afraid the short duration of the history points to a malignant tumor, and the signs of bilateral involvement indicate that the patient may have an intrinsic lesion, a glioma of the brain stem.

To my way of thinking glioma of the brain stem is the most likely diagnosis. I do not know of any thrombotic occlusion of any branch of the vertebral or basilar artery, or even of several branches, that would be likely to give such a striking lateralization involving the cranial nerves with so little involvement of the long tracts going down to the limbs and torso, so I should be inclined to exclude that and favor the intrinsic lesion, a glioma of the brain stem, which tends oddly enough to spare the long tracts in the early phases of growth. Nevertheless, I should strongly urge the course that was apparently carried out, that of an operation.

Clinical Diagnoses

Tumor of posterior fossa.
Infarct, left cerebral hemisphere, old.

Dr. William H. Sweet's Diagnoses

Glioma of brain stem.
Multiple vascular lesions, cerebral hemispheres.

Anatomical Diagnoses

Glioma, malignant, unclassified, cerebellum and brain stem, with leptomeningeal invasion.
Infarcts, embolic, old, left temporal and occipital lobes.

Pathological Discussion

Dr. Edgar B. Taft: Dr. Ballantine, you saw this patient in the hospital.

Dr. H. Thomas Ballantine: Dr. Sweet is to be congratulated on his analysis of this situation, particularly since we withheld the air studies from him.

The first operation performed on this patient was a pneumoencephalogram, which showed a filling defect of the fourth ventricle on the right consistent with a neoplasm of the posterior fossa. Consequently, a cerebellar exploration was carried out and on inspection of the posterior fossa it was seen that the fifth, seventh and eighth nerves were markedly thickened, actually almost curled on themselves, in such fashion that they seemed to be jammed into their foramens of exit; particularly was that true of the seventh and eighth nerves. When the cerebellar hemisphere on the right was explored, a grayish, more or less gelatinous tumor mass was found that on frozen section was said to be metastatic carcinoma, and a resection of the portion of the tumor occupying the right cerebellar hemisphere was carried out. From the peculiar appearance of the cranial nerves, we thought that they too were invaded by tumor as well as perhaps the brain stem. This patient did fairly well for about ten days after the biopsy and then went slowly downhill with further cranial-nerve involvement and died twenty-five days after operation.

Dr. Taft: The surgical specimen that we received caused considerable comment. The opinions varied between lymphoma, medulloblastoma and metastatic undifferentiated carcinoma. The specimen was finally signed out with the last diagnosis. The tumor was composed of small round cells with not too large nuclei and scanty, but usually fairly well defined, cytoplasm. The chromatin was finely divided; some cells contained very prominent nucleoli, and mitoses were quite frequent. The tumor did not seem to be growing in any particular pattern.

There is a note in the record that the patient was treated with x-ray therapy. Do you know whether it had any beneficial effect?

DR. BALLANTINE: None whatever. After the tenth postoperative day since the course was poor, x-ray therapy was given as a trial to the posterior fossa from which the tumor had not been completely removed.

DR. TAFT: At autopsy this patient was carefully examined for a primary tumor site. The lungs, gastrointestinal tract, kidneys and other organs were unremarkable. In the heart there was minimal rheumatic involvement of the mitral valve. It was thought that the embolic phenomena involving the brain that had been noted clinically had probably arisen from the left atrial appendage although at the time of autopsy there was no thrombus present there. In addition to infarcts in the brain, there were 20 to 25 infarcts up to 2 cm. in diameter in the kidneys. There were no infarcts in the spleen although it was very small at the time of autopsy, weighing only 40 gm.

The brain will be described by Dr. Richardson.

DR. RICHARDSON: I should like to add my congratulations to Dr. Sweet for his fine discussion of this case.

On examination of the brain we found lesions of two sorts, vascular and neoplastic. The cerebral vessels were almost entirely free of atherosclerosis. There was a large lesion typical of old infarction in the left cerebral hemisphere involving the left superior temporal gyrus throughout its entire extent. There was a similar large lesion on the inferomedial surface of the left occipital lobe. This involved the fusiform gyrus and the medial aspect of the occipital area to within approximately 2 cm. of the occipital pole, extending into the white matter sufficiently to encroach on the optic radiations.

In the right cerebellopontine angle, a lesion of an entirely different sort was encountered. In this region cerebellar tissue had been removed at operation. The edges and base of the operative defect in the cerebellum, the neighboring leptomeninges and the right side of the pons were infiltrated with homogeneous, whitish tumor tissue. This lesion was extremely difficult to classify. The cells were as described by Dr. Taft. The tumor was invasive and infiltrating, without well defined borders. Its greatest mass was in the right cerebellar hemisphere and middle cerebellar peduncle, but it extended into the leptomeninges and into the seventh and eighth cranial nerves. Glial stains showed that these cells were not elaborating glial fibers. On the other hand, there was not an extensive collagenous or reticular matrix. The cells themselves, and their arrangement, did not suggest an epithelial tumor. Because of its growth characteristics and staining properties we believe that it most probably was a very malignant undifferentiated glioma, but we have not been able to classify it more exactly.

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CASE 39492

PRESENTATION OF CASE

A twenty-eight-year-old divorced waitress entered the hospital because of severe pain in the right-flank region.

She was well until three weeks before admission, when she went on a diet during which she noticed some fatigue. Two days before admission moderately painful cramps began in the right lumbar region that she attributed to the onset of her period. The pain left, and she slept well but was sluggish and tired the next day and remained home from work. There was no nausea or vomiting, no burning on urination and no residual pain in the right side. At about 5 p.m. she felt chilly and shivered but did not take her temperature. She went to bed and again slept well. On the day of admission she had a good breakfast but felt tired. At about 1 p.m. she had an attack of severe, sharp and colicky pain in the right flank that radiated to the lower abdomen and right thigh. She vomited three times and was not relieved by changes in position or by Empirin. Thirty minutes later she passed a normal stool. She continued to feel hot and had mild chills. There was urinary frequency but no dysuria or obvious change in the color of the urine.

The past history included an appendectomy at the age of sixteen years followed by an abscess that had to be drained. The menarche occurred at the age of fourteen, with periods every twenty-seven to twenty-nine days that lasted four to five days. She had pain in the lower back during the first day or two of each period. The last menstrual period started the day of admission, twenty-eight days after the previous period. There were no children by the marriage that had ended years before. She had been having regular sexual intercourse in the recent past.

Physical examination revealed an obese woman weighing 170 pounds who was in moderate distress. The eyes, nose and throat were normal, and the lungs were clear. The heart was normal, with no murmurs. Examination of the breasts was negative except for slightly enlarged nipples. The abdomen was soft but tender to palpation over McBurney's point, with slight tenderness away from this area. There was no rebound tenderness or spasm, and peristalsis was normal. Contraction of the psoas muscle was painful. There was marked tenderness

in the right costovertebral angle and slight tenderness on the left. Pelvic examination was deferred because of the menstrual period.

The temperature was 100°F., the pulse 84, and the blood pressure 105 systolic, 80 diastolic.

The urine had a specific gravity of 1.025, and was negative for bile, albumin and sugar. The sediment contained occasional red cells and white cells per high-power field. The hemoglobin was 14.5 gm. per 100 cc., and the white-cell count 21,000. A stool was guaiac negative. A plain film of the abdomen was normal.

Penicillin and streptomycin were started, and the next morning she felt somewhat better but had constipation because straining led to pain. She had "pain all over" and complained of pain under the right leaf of the diaphragm and on the right shoulder. The abdomen was soft, with slight rebound tenderness in the right lower quadrant. Pelvic examination showed tenderness in the cul-de-sac, tenderness in the vaults, more on the right than on the left, and a "clean" cervix. The uterus was not outlined because of obesity. No masses were felt. The period continued and was not remarkable. The temperature fell to 98.6°F., and a second white-cell count was 14,900, with 80 per cent neutrophils. Another urinary sediment contained 1 to 3 red cells per high-power field. On the fourth hospital day she had a few crampy pains in the lower abdomen, voluntary guarding but no true spasm and a normal temperature. The period had stopped, and there was a minimal whitish discharge. Her condition remained the same, and on the sixth hospital day an operation was performed.

Differential Diagnosis

Dr. Charles G. Mixter, Jr.*: I wonder if we could see the x-ray film first.

Dr. Joseph Hanelin: On the day of admission a plain film of the abdomen showed no remarkable findings. In the intravenous pyelogram done the following day the urinary tracts are grossly normal, and the bladder shadow is essentially normal. However, there is a suggestion of an abnormal soft-tissue density lying above the bladder that looks larger than a normal-sized uterus (Fig. 1). There is no visible calcification within the density itself. No ureteral obstruction is seen.

Dr. Mixter: The normal pyelogram helps me. This patient appears to have been suffering from an inflammatory or an acute ischemic process that gave rise to symptoms and signs on the right side of the abdomen. Apparently, this was initiated by an acute attack, which subsided, then recurred and seemed to be subsiding at the time of the operation. I am at a loss to understand why an operation was undertaken at the time that the patient was asymptomatic. The temperature had fallen to normal, the white-cell count apparently was falling, and there was no evidence of any localizing signs except possibly the mass seen in the x-ray film but not mentioned in the protocol.

In the time I have I cannot enter into a detailed discussion of all the possibilities. From the history that is given, any organ on the right side of the body from the diaphragm to the pelvis within or close to the peritoneal cavity could be implicated; therefore, I shall start with the genitourinary tract. The first processes that come to mind are infection and obstruction. Obstruction could have been due to either a stone or a congenital abnormality and

Figure 1. *Roentgenogram of the Abdomen.*
Arrows point to the abnormal soft-tissue density within the bony pelvis lying above the urinary bladder.

would be consistent with a story of flank pain radiating down to the lower abdomen and into the thigh and accompanied by a few red cells in the urine. It does not, however, quite fit in with the picture seen in the second x-ray films, nor does it give us much clue to the reason for the pelvic findings at a later time. I believe we can pass pyelitis over except as an accompaniment of an obstruction. A ruptured cyst or tumor of the kidney seems unlikely although such a condition might give rise to psoas-muscle signs and possibly some extravasation down to the pelvis. I might ask Dr. Hanelin whether or not the mass that is seen on the second film was seen on the first one.

Dr. Hanelin: It is difficult to see on the original film.

Dr. Mixter: Could this be an extravasation from the ureter?

Dr. Hanelin: It could be a fluid collection; it does not have to represent a solid or cystic tumor.

*Assistant in surgery, Massachusetts General Hospital.

DR. MIXTER: Could it be extravasation of the dye?

DR. HANELIN: No; I think the dye is well localized to the bladder.

DR. MIXTER: Turning to gynecologic problems, one of the first things one thinks of in a patient of this age and with this story is acute pelvic inflammatory disease, which is often accentuated at the time of the menstrual period. In favor of this diagnosis is the story of rapid subsidence under antibiotics. It is unfortunate in two respects that no smear was made of the so-called discharge: it might have been of importance to have found out whether there were gram-negative diplococci.

Another possibility is an ectopic pregnancy. Admittedly, this would have been unusually early for an ectopic pregnancy to manifest itself, but the attack of pain with eventual signs of diaphragmatic irritation, possibly peritoneal irritation, and tenderness on pelvic examination fit in with that picture. A rapid test for pregnancy would have ruled that in or out. In view of the relatively normal period coming on at essentially the proper time, I believe I can rule that out. Other studies that could have been done to establish this diagnosis would have been to follow the red-cell count and the hemoglobin, which was not mentioned, for evidence of a slow blood loss or a more rapid one, though apparently from the history there was no other evidence for that.

Another condition that I might consider is an ovarian cyst. Certainly an ovarian cyst in an obese patient might readily be missed. An ovarian cyst could have twisted, become ischemic, untwisted and then recurred with further ischemia and possibly rupture, releasing fluid in the abdomen that irritated the diaphragm. I think this is an excellent possibility.

In the gastrointestinal tract a few conditions might be considered. One is acute appendicitis. I say that with the full realization that an appendectomy had previously been performed; however, appendicitis occasionally occurs in a part left behind at a previous appendectomy. The history of pain over McBurney's point is fairly consistent with such a diagnosis but does not explain the diaphragmatic pain unless the pelvic mass represented an abscess due to a ruptured appendix. I do not think an ulcer in the gastrointestinal tract would have given this kind of history although perforation of an ulcer could have caused the collection of fluid in the pelvis. There was no mention of air under the diaphragm. I think I can rule out such an ulcer as well as ulcerative colitis and regional enteritis, all of which should have given previous symptoms. Diverticula, which occur in the duodenum and small bowel, may rupture, producing pain, an elevated temperature and evidence of peritoneal irritation. Although these are possibilities, they are rare. Of course, one should also consider a Meckel diverticulum and diverticula in the ascending colon, which again quite rarely give symptoms. There is little evidence for intestinal obstruction. Another condition that might occur in the small bowel and produce a picture like this is a localized mesenteric thrombosis, but in a patient of this age with no previous history of cardiac disease and guaiac-negative stools, I should certainly not think of this very seriously.

Although there is a chance that this patient had biliary-tract disease, such as acute cholecystitis with rupture and peritonitis, I should expect that she would have been much sicker and that the attack would not have subsided as fast as it did. There would have been a past history of gall-bladder disease. Rupture of a liver cyst or a spontaneous rupture of the liver, which are both extremely rare, I suppose could give a history somewhat like this, but without evidence of shock and a falling red-cell count I cannot substantiate either. Pancreatitis certainly may be initiated with pain in the costovertebral angle and back, as well as in the flanks, but the rest of the picture is hardly consistent with it and an amylase determination was not done.

Because of the few symptoms and with so little evidence of disease, I think I should stick to the statistically more frequent conditions and shall pick as my first diagnosis an ovarian cyst, with torsion and possibly rupture.

DR. WELDON W. WEST: Pelvic examination on the day of entry revealed an acutely tender mass in the right vault of the pelvis. The lack of free peritoneal fluid and the absence of evidence for significant blood loss made us consider an acute tubo-ovarian abscess as the most likely diagnosis. Laparotomy was done because in the face of rapid subsidence of acute symptoms, both locally and systemically, the mass persisted. We did not consider a twisted or infarcted cyst because of the adherence of the mass.

CLINICAL DIAGNOSIS

Tubo-ovarian abscess.

DR. CHARLES G. MIXTER, JR.'S DIAGNOSIS

Twisted ovarian cyst, with ? rupture.

ANATOMICAL DIAGNOSIS

Tubal pregnancy.

PATHOLOGICAL DISCUSSION

DR. WEST: Laparotomy revealed the greater bulk of the mass to be omentum that had wrapped itself about the right tube and ovary. With release of the omentum, free bleeding occurred from the tube, and a blood clot containing an amniotic sac and fetus was found inferior to the tube. A right salpingectomy was done. There was no generalized peritoneal reaction.

DR. EDGAR B. TAFT: We could not tell from the operative specimen whether or not the tube was ruptured at the time of operation. In the laboratory we received a mass of blood clot with an intact amniotic sac containing a 1-cm. fetus. The tube submitted also contained some blood clot and remnants of placental tissue. Uterine curettings showed typical decidual reaction.

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WELLCOME CENTENNIAL

THE hundredth anniversary of the birth of Sir Henry Wellcome coincided almost exactly with the opening of the First World Conference on Medical Education in London on August 24, 1953. Consequently, the centennial exhibition of Sir Henry's life and works on display at the Wellcome Research Institution within a block of Friends' House, where the principal meetings of the Conference were held, attracted considerable attention.

Of special interest to Americans is the fact that Henry Wellcome was one of those unusually intrepid fellow countrymen who ignored Horace Greeley's famous injunction to the young man to go where the course of empire took its westward way. Putting the reverse English on this popular slogan in a truly literal sense he retraced eastward his ancestors' footsteps to find fortune and eventually fame in England.

Henry Solomon Wellcome was born at Almond, Wisconsin, on August 21, 1853. His father, although raised as a farmer, moved to Garden City, Minnesota, during Henry's youth, and eventually took over his brother's drugstore in that town. At the age of seventeen Henry took a position as pharmacist's clerk in Rochester, leaving two years later to study pharmacy in Chicago and later in Philadelphia. The year 1876 found him traveling extensively for the drug firm of McKesson and Robbins of New York.

Soon another enterprising young American, Silas M. Burroughs, London agent for the Philadelphia firm of John Wyeth and Brother, urgently invited Wellcome to join him in partnership in London, with a view to introducing to Europe American methods of manufacturing compressed medicines. With his firm's blessing Wellcome accepted and in 1880 sailed for England carrying with him not only the blessing but the exclusive agency for McKesson and Robbins' products "for Europe, Asia, Africa, the East Indies and Australia."

Hard work and imagination rapidly built the partnership into one of the world's large pharmaceutical industries. In 1894 the Wellcome Physiological Research Laboratories were established for the manufacture of diphtheria antitoxin and for long-term research. In 1895 Burroughs died, and Henry Wellcome assumed the entire management of the business; in 1896 the Wellcome Chemical Research Laboratories were developed, in 1901 the Wellcome Tropical Research Laboratories were opened at Khartoum, Egypt, and in 1913 the Wellcome Bureau of Scientific Research in London was established for the study of tropical disease.

The character and continuance of the business assured, Henry Wellcome, always interested in cultural pursuits, in scientific and literary collecting and in humanitarianism, gave more and more of his time to such activities. He became interested in archaeology and not only supported expeditions but himself organized and led one in Egypt. Here, disturbed by the lawlessness and intemperance of the native workers employed in the excavating, he organized an Order of the Peacock Feather, cere-

moniously awarding one of these ornithologic specimens to any worker who abstained from ardent spirits for an eight-week period. So attractive was the inducement that a rush order had to be sent to England for a consignment of peacocks to supply the coveted prizes.

Early in his career Wellcome had started a collection of objects and texts associated with the history of medicine and the allied sciences. From this accumulation the Wellcome Historical Museum and its Library, now housed in the imposing Wellcome Research Institution in Euston Road, developed. Here in the Museum are over 200,000 major exhibits, and in the Library more than 200,000 volumes, 10,000 manuscripts and over 100,000 autograph letters. These include 612 complete works and 22 fragments in incunabula.

Wellcome's major honors came to him late in life. In 1928, when he was nearly seventy-five, the University of Edinburgh conferred on him the honorary degree of Doctor of Laws. In 1932 he received a knighthood from King George V and in the same year was made an honorary fellow of the Royal College of Surgeons and a Fellow of the Royal Society.

He died in 1936; under the terms of his will all the profits of the Wellcome Foundation Ltd. declared as dividends were thereafter to go to "the advancement of research work bearing upon medicine, surgery, chemistry, physiology, bacteriology, therapeutics, materia medica, pharmacy and allied subjects." The establishment or endowment of research museums or libraries and for the collection of information connected with the history of medicine and its allied subjects was also provided for. This is said to be the first instance in Britain whereby the profits from a commercial firm "are permanently dedicated to the advancement of knowledge for the benefit of mankind."

PILONIDAL COMPLEX

THE account by Dwight and Maloy of their experience with pilonidal sinus published elsewhere in this issue of the *Journal* will be of value to all who have struggled with this lesion. Dwight's long interest and vast experience render him an expert among experts, and it is significant that he concludes that the simplest measures in either closed or open operations are the best.

The first known account of what was probably a pilonidal sinus may be found in a letter by A. W. Anderson, of Gray, Maine, published in the *Boston Medical and Surgical Journal* of February 8, 1847. Dr. J. Mason Warren, of Boston, described a similar lesion as "an abscess containing hair on the nates" in 1854, but it was not until 1880 that R. M. Hodges, again in the *Boston Medical and Surgical Journal*, coined the term "pilonidal sinus." Hodges gave an excellent description of the lesion and postulated that the following elements are necessary for its development: "A. The presence of a congenital coccygeal dimple. B. Abundant pilous development: hence adult age and almost of necessity the male sex. C. Insufficient attention to cleanliness: consequently, subjects, as a rule, must be persons of the lower class, and the infection one met with in hospital, or dispensary, rather than private practice." Regarding treatment, Hodges wrote, "If it is gently opened and the lock of hair removed, the sinus fills up with new tissue and in due time heals by solid cicatrix." Anderson also noted that the abscess healed promptly once the hair was extracted. In this issue of the *Journal*, after a hundred and six years and no one knows how many hundred thousand operations, papers and new operative technics, about the same conclusion seems to have been reached, — namely, that if the abscess is opened gently and the hair removed it will heal "in due time."

It is the interpretation of what constitutes "due time" that has occasioned so much controversy and confusion and, in the hands of the casual operator employing a new technic, so many unfortunate and bad results.

All methods of dealing with a pilonidal sinus have in common the grim determination of their originator to make them more effective, and, by dint of exceptional care with the operation or, in open operations, with the dressings, better results are obtained. It is extraordinary that so simple a lesion should tax the surgeon's ingenuity, but it is about time to admit the fact that what it taxes is not the adequacy of the method employed but

how well, how carefully and how gently the surgeon executes it. It is probable that if all operations on pilonidal sinuses were performed only by neurosurgeons who are accustomed to handling tissues with the gentlest technic, the results would be vastly improved and primary closure in selected cases would be invariably successful.

It is obvious, however, that this little lesion has not yet been mastered. Even Dwight, despite his extensive experience and his evident satisfaction with simple methods of management, cannot refrain from describing a new method of primary closure employing a suture that "occasionally breaks when it is removed" and, according to his own figures, appears to have no advantage over excision with ordinary suturing. It is unlikely that the matter can end on this note, and the next report on this subject is calmly awaited. This will undoubtedly insist that if the Militzer stitch as described by Dwight is passed from left to right, as a left-handed operator would pass it, and if the suture material contains 0.0018 microgm. of vanadium, the cases in which it breaks when pulled out will be reduced by 4.5 per cent.

ANTIEMETIC DRUGS IN THERAPY

Nausea and vomiting constitute one of the most common symptom complexes observed in medicine. They can be most distressing and, at times, definitely harmful. Fortunately, most cases of nausea and vomiting are easily controlled or of short duration. The common occurrence of these symptoms after the use of morphine, meperedine (Demerol), cardiac glycosides and many other drugs is exhausting to the patient and can be so severe as to prevent the proper use of these highly effective agents. The nausea and vomiting of pregnancy, although usually not a serious problem, can be trying to both patient and physician. When vomiting persists, the dehydration, lack of nourishment and electrolyte disturbance so created present a serious problem. This is particularly true of the severe vomiting observed in carcinomatosis and uremia and of pernicious vomiting of pregnancy, in which the usual therapeutic measures are not very successful.

The recent announcement of a new drug useful in the suppression of this symptom complex is of considerable interest.* This substance, known experimentally as 2601-A, is a chlorinated phenothiazine and a close relative of the antihistaminic (Phenergan). However, it has no antihistaminic action. It effectively suppresses the emetic effect of apomorphine in dogs. Preliminary studies showed it to be effective in patients who have nausea and vomiting caused by carcinomatosis, uremia and pregnancy and by the use of other drugs such as morphine. The relief is usually prompt and striking, and maintained as long as the drug is continued or until the underlying cause is corrected. Patients relax and are able to take nourishment by mouth — sometimes the first food they have been able to eat in weeks.

Not all patients receive complete relief from this medication, some becoming drowsy or being made restless by it, but the high degree of effectiveness and low degree of toxicity indicate that 2601-A is likely to be a valuable addition to the therapeutic agents.

A word of caution is necessary as with nearly any new, highly effective drug. The use of an antiemetic such as 2601-A is symptomatic therapy; therefore, the physician must make certain of the mechanism before suppressing the indication of underlying disease. As one should not give morphine for an abdominal pain of uncertain etiology, neither should one give an effective antiemetic until the cause of the nausea and vomiting is ascertained and these valuable warning signs are no longer needed.

*Fried, D. G., and Cummins, J. F. New antiemetic drug. *J. A. M. A.* 153:460, 1953.

DISTINGUISHED SERVICE

The Columbia-Presbyterian Medical Center in New York celebrated in October its twenty-fifth anniversary by conferring on twenty-five recipients the first Distinguished Service Awards ever to be made by the Center.

All the recipients had been associated with the institution for many years; all of them, representing the major branches of medicine, dentistry and nursing, had made distinguished contributions to the growth and development of the Center. Statistically, the recipients of the new honors have totaled

more than eleven hundred years of service to medical science; their average term of service, mostly as professors in the College of Physicians and Surgeons and as consultants at the Presbyterian Hospital, has been forty-five years — much of it, obviously, before the Center was established as such.

The group contains 24 men and 1 woman — Miss Helen Young, director emeritus of the School of Nursing. Miss Young, who served as head of the School from 1921 to 1933 entered it as a probationer in 1909, after having already been a public-school teacher for over ten years. In 1937 she was awarded the University Medal for Excellence for her service to Columbia of more than twenty-five years.

Not to be taken lightly are such symbols of distinction in the various walks of life — mute testimonials that represent the cogent words: "Well done!"

The profession has been served with a pamphlet containing proofs and evidences of the purity and medical properties of Wolfe's Schiedam Aromatic Schnapps.

Boston M. & S. J., *December 7, 1853*

MASSACHUSETTS MEDICAL SOCIETY

DEATHS

O'Sullivan — Anna O'Sullivan, M.D., of Boston, died on November 18. She was in her seventy-eighth year.

Dr. O'Sullivan received her degree from Tufts College Medical School in 1910. She was a fellow of the American Medical Association and was formerly secretary of the Massachusetts Women's Medical Society.

Payne — Edward A. Payne, M.D., of Woburn, died on October 26. He was in his forty-third year.

Dr. Payne received his degree from Tufts College Medical School in 1933. He was a member of the staff of Choate Memorial Hospital.

He is survived by his widow, a daughter, three sisters and two brothers.

CORRESPONDENCE

REVOCATION OF LICENSE

To the Editor: At the meeting of the Board of Registration in Medicine held October 22, 1953, it was voted to revoke the registration of Dr. Gil J. Cordero, 17 Woodworth Avenue, Springfield.

Robert C. Cochrane, M.D.
Secretary

State House
Boston

To the Editor: At the meeting of the Board of Registration in Medicine held October 22, 1953, it was voted to revoke the registration of Dr. Jean C. Marchand, Linden Street, Salem.

Robert C. Cochrane, M.D.
Secretary

State House
Boston

REGARDING MYRINGOTOMY

To the Editor: In the *Journal* of October 15, 1953 an editorial entitled "Otitis Media and Myringotomy" based on a paper by Dr. Zonderman, suggests that the opinion of the editorial staff is that adequate antibiotic treatment has made routine myringotomy unnecessary in acute otitis media. The impression made on the average reader may well be that the editors have said that myringotomy has been rendered obsolete by proper antibiotic medication.

The obvious purpose of Dr. Zonderman's article is to improve the treatment of acute otitis media so that there will be fewer sequels of deafened and damaged ears. The author makes a plea for examination of the eardrums. Unless this is done the indications for myringotomy are often not apparent in the patient receiving antibiotics. When the myringotomy is *indicated* Dr. Zonderman stresses that it should be done to provide surgical drainage of pus, to obtain a culture and to prevent the sequels of serous otitis media, chronic otitis media, surgical mastoiditis and — above all — a deafened ear.

Because the author omitted the words "when indicated" after "a properly performed myringotomy," the editorial writer took the phrase to mean that myringotomy *whether indicated or not* should be carried out as a routine procedure. Routine myringotomy in acute otitis media is not necessary, as anyone who has used the antibiotic drugs well knows. But myringotomy when indicated is one of the important therapeutic measures that should not be omitted. It is a safe, conservative and simple procedure. It is in no way to be thought of as opposed to antibiotic therapy, but is to be employed in conjunction with antibiotic measures.

The *Journal* must support conservation of hearing. I suggest that the editorial board emphasize the point that "a deafened ear, even though bacteriologically cured, represents a therapeutic failure." I fear that the editorial "Otitis Media and Myringotomy," belittling routine myringotomy, is apt to result in an increase in the number of cases of unnecessary deafness.

John R. Richardson, M.D.

Boston

Note: The editorial was intended to clarify unequivocally the point made by Dr. Richardson in the third paragraph of his letter, that routine myringotomy in acute otitis media is not necessary; the importance of myringotomy when indicated was stressed in Dr. Zonderman's paper. It is doubted if any reader of both the article and the editorial would reach the entirely unwarranted conclusion that myringotomy has been rendered obsolete by proper antibiotic medication or that eardrums should not be examined. Certainly a deafened ear, even though bacteriologically cured, represents a therapeutic failure. The opinion was merely expressed editorially that the relative importance of antibiotics and myringotomy warrants further evaluation by a suitably planned series of cases. — Ed.

RESTORATION OF FUNDS TO VETERANS ADMINISTRATION

To the Editor: Too little publicity has been given to the restoration of funds to the Veterans Administration in payment of the costs for treatment of veterans with service-connected disabilities. For this reason, many veterans and doctors holding fee appointments with that agency are of the opinion that treatment — unless during an emergency — will not be authorized.

The truth of the matter is that almost all the funds for this purpose, which were withheld temporarily, have been released to the Veterans Administration. Therefore, the local authorities of the Veterans Administration are financially able again to authorize treatment that, although not emergency, is needed and desired. It appears quite evident that the Veterans Administration will not publicize the fact that more money is now available. Consequently, the reauthorization of treatment for many veterans is dependent upon the action of individual fee-basis physicians.

Humphrey L. McCarthy, M.D., *Chairman*
Committee on Veterans Affairs
Massachusetts Medical Society

BOOK REVIEWS

Free and Unequal: The biological basis of individual liberty. By Roger K. Williams. 8°, cloth, 177 pp., with illustrations. Austin: University of Texas Press, 1953. $3.50.

This is the type of book that one reads not to discover something new but to find a new approach to a problem with which most physicians are familiar. The author has collected and classified the many ways in which people differ from each other biologically. He has drawn certain conclusions, examined certain problems and made some definite proposals. No one can very well dispute his facts, but many will not agree that the problems involved are as serious as he believes, and many more will not agree with the possible solutions he suggests.

The following quotations are considered typical: "Human worth resides not only in those whom we regard as great but in all of us, and we should provide an environment which will give everyone an equal chance to develop his potentialities in the way best suited to him individually." "One of the prime functions of this book is to examine various areas of life to see where distinctiveness plays an important part and where it does not." "It is obvious that there are many kinds of laws which, because of fundamental differences in people, could not be enforced if we had any reason to want to enforce them." "The diversity of men's minds, coupled with the tacit ignoring of this diversity, is the fundamental basis of hatred."

The volume should be read by all people interested in education and those interested in the study or treatment of almost any aspect of mankind. This stimulating, informative and thought-provoking study has a place among attempts to understand the wide variety of present-day problems.

WARREN BURNS, M.D.

Group Psychotherapy: Studies in methodology of research and therapy. Report of a Group Psychotherapy Research Project of the U. S. Veterans Administration. By Florence B. Powdermaker, Ph.D., M.D., and Jerome D. Frank, Ph.D., M.D. 8°, cloth, 615 pp., with 32 tables and 7 charts. Cambridge: Harvard University Press, 1953. $6.50.

This volume represents a progress report of a research project in group psychotherapy carried out over a period of approximately two years (1947-49) by the Veterans Administration under the auspices of the Washington School of Psychiatry. After the war the Veterans Administration, like many organizations, was faced with the great disproportion between the number of mentally disturbed persons needing care and the relatively few psychiatric workers available. Group psychotherapy had become an established psychiatric method by this time, although not as yet well documented except by a handful of workers, and so it seemed natural to give it more attention in the hope that it could solve the ever-increasing problem of the emotionally ill. The present investigation concentrated on two classes of psychiatric illness: the neuroses present in those coming to the mental-hygiene clinic, and the schizophrenic patients currently hospitalized who had not responded to prolonged hospitalization or to electroshock therapy.

The book is about equally divided to correspond to the two projects and consists of a detailed analysis of almost every aspect of the problem, the magnitude of which may be judged from a few of the subjects considered: group composition, early meetings, group functioning at later stages, hostility toward the doctor, antagonism between group members, resolving tension and prolonged silences. It is regretted that no comprehensive conclusions are made although the authors may consider them better omitted in a "progress report."

Of particular interest is the authors' appraisal of the roles of the physicians in this project. The psychiatrists had greatly varying personalities, training and experience in this work, and some even had no previous experience with group psychotherapy. Within limits it was found that training and experience were not too important but that the major personality traits and personal problems of the psychiatrists had a profound influence on their handling of certain groups — for example, whether they were directive or nondirective and how they handled the group's hostility to them. In both the neurotic and the schizophrenic groups the best results were obtained by the most experienced psychiatrists, but when the younger, inexperienced psychiatrists overcame their initial anxieties and recognized the effect of their personal problems on the situation, better relations and results were obtained.

For the student of group psychotherapy the study should be highly rewarding. All in all, there is a wealth of material here, and from the point of view of research in group therapy this will prove to be an important reference work for some years to come. A serious omission is the failure to discuss the question of the termination of therapy.

Clinicians more interested in results than methodology will tend to be disappointed in this volume. The schizophrenic group of approximately 100 patients fared only slightly better than the control group. Only a few cases (about 6) could really be called therapeutic successes, and the authors frankly doubted if the patients had any insight in the traditional sense; nevertheless, it was believed that there was a slight, but definite, therapeutic effect, which allowed the experimental group to gain more in the way of improved ward behavior, gate passes and ground privileges. It is interesting that the experimental group required more daytime sedation, but less nocturnal sedation than the controls. In this study the patients who became disturbed were temporarily taken out of the group, given electric shock, packs and so forth and returned when their behavior was no longer disturbing. In the second year of the project the frequency of packs, tubs, electric shock and "guest days in the acute service" was appreciably reduced in the experimental group, but peculiarly enough this also occurred in the control group, although to a lesser extent.

The neurotic group consisted of 124 patients, 24 of whom received group therapy alone, and the rest combined group and individual psychotherapy. It must have been distressing to the workers to record that 74 patients left the groups, although 31 continued in individual therapy. However, 75, or 61 per cent, were considered to have shown some degree of improvement, although only 10 per cent of these had manifested gains in all three "areas" of possible change described by the authors. It may be estimated from some of the statistics and by inference that between 1000 and 1500 hours were spent on this group of patients by several members of the research team, and in spite of the exceptional nature of the study, one begins to wonder if group therapy was indeed the answer to the problem that prompted this research and confronts all in the field. The 13,349 patient hours spent on the schizophrenic group seems a frightening waste of time for such meager rewards, although there must be many who wonder why this particular group was chosen in preference to patients recently admitted.

It is clear from this study that group psychotherapy is not the definitive solution to the problem, but it is hoped that such studies will soon establish certain technics, indications and contraindications that will place it properly in the field as an adjuvant in the comprehensive attack on the problems presented by the mentally ill.

IAN HAY BROWN, M.D.

BOOKS RECEIVED

The receipt of the following books is acknowledged, and this listing must be regarded as a sufficient return for the courtesy of the sender. Books that appear to be of particular interest will be reviewed as space permits. Additional information in regard to all listed books will be gladly furnished on request.

Current Problems in Psychiatric Diagnosis. Edited by Paul H. Hoch, M.D., New York State Psychiatric Institute, and Columbia University College of Physicians and Surgeons; and Joseph Zubin, Ph.D., New York State Psychiatric Institute, and Department of Psychology, Columbia University. 8°, cloth, 291 pp. New York: Grune and Stratton, 1953. $5.50. *(Proceedings of the Forty-First Annual Meeting of the American Psychopathological Association, held in Philadelphia, Pennsylvania, June 1951.)*

This volume presents the papers read at the meeting by 25 persons. The material is divided into five main parts on perspectives on diagnosis and etiology, prognosis in relation to diagnosis and etiology, cultural and social factors in diagnosis and etiology of mental disorders, and current trends in diagnosis and etiology. The presidential address, "The Responsibility of Psychiatry," by Dr. Lauren H. Smith, forms the fifth part. There is a good index. The volume as a serial should be in all medical libraries and available to psychiatrists.

Nervous Transmission. By Ichiji Tasaki, M.D., Central Institute for the Deaf, St. Louis, Missouri. 8°, cloth, 164 pp., with 77 illustrations. Springfield, Illinois: Charles C Thomas, 1953. $7.50.

Dr. Tasaki began his work on nerve and muscle in 1935. In this monograph he presents the results of his work on nervous transmission and discusses the effects of refractoriness, narcosis and electrotonus. The last chapter gives an account of the properties of small myelinated and nonmyelinated nerve fibers. There is a bibliography of 7 pages. The monograph should be in collections on physiology and on the nervous system.

Diagnostic Tests in Neurology: A selection for office use. By Robert Wartenberg, M.D. With forewords by Sir Gordon Holmes, M.D., F.R.S., and Stanley Truman, M.D. 8°, cloth, 228 pp. with 61 illustrations. Chicago: Year Book Publishers, Incorporated, 1953. $4.50.

This manual is written primarily for the general practitioner as a supplement to the textbooks on neurology. The material is limited exclusively to clinical tests. The whole of the nervous system is covered, and there is a special index for the pertinent tests of morbid conditions. There is also a general index.

The Metabolism of the Tubercle Bacillus. By William F. Drea, D.M.D. associate laboratory director, Colorado Foundation for Research in Tuberculosis, Colorado College, Colorado Springs, Colorado; and Anatole Andrejew, I.C., research staff, National Center for Scientific Research in France, Pasteur Institute, Paris. With a foreword by Esmond R. Long, M.D., Ph.D., Sc.D., director and professor of pathology, University of Pennsylvania School of Medicine; and director of medical research, National Turberculosis Association, Philadelphia. 8°, cloth, 448 pp., with 78 tables. Springfield, Illinois: Charles C Thomas, 1953. $12.50.

This monograph surveys the pertinent literature on the metabolism of the tubercle bacillus. The text is divided into 5 chapters on nutrition and growth, depth growth, respiratory exchanges, enzymes and products of metabolism. There is a bibliography of 50 pages and a good index. The book should be in all medical libraries and in collections on tuberculosis, bacteriology and biochemistry.

The Pluripotency of the Hypophyseal Hormones and Consequences for Endocrinology and Cancerology. By Jules Samuels, specialist for endogenous endocrinotherapy and director, Central Institution for Samuels-Therapy, Amsterdam. 8°, cloth, 296 pp. Amsterdam, Holland: Ed. N.V. Cycloscoop, 1953. Bound, 37 guilders.

Dr. Samuels, in this new book on the hypophyseal hormones, divides his text into 3 parts on the cell-dividing and retarding factors, cell-enlarging and accelerating factors, and the significance of the pluripotent qualities of the pituitary hormones for oncology, in particular for cancerology. The author surveys the literature, and there is a bibliography of 56 pages.

Atlas of Tumor Pathology, Washington, 1952. Prepared at the Armed Forces Institute of Pathology. (May be obtained from the American Registry of Pathology of the Institute.)
Fascicle 14. *Tumors of the Thyroid Gland.* By Shields Warren, M.D., and William A. Meissner, M.D. 4°, paper, 97 pp., with 31 plates. $1.75.
Fascicle 15. *Tumors of the Parathyroid Glands.* By Benjamin Castleman, M.D. 4°, paper, 74 pp., with 30 plates. $0.65.
Fascicle 17. *Tumors of the Lower Respiratory Tract.* By Averill A. Liebow, M.D. 4°, paper, 189 pp., with 73 plates. $1.25.
Fascicle 31b and 32. *Tumors of the Male Sex Organs.* By Frank L. Dixon, M.D., and Robert A. Moore, M.D. 4°, paper, 179 pp., with 6 plates. $1.50.
Fascicle 35 and 37. *Tumors of the Central Nervous System.* By James W. Kernohan, M.D., and George P. Sayer, M.D. 4°, paper, 129 pp., with 45 plates. $0.90.

This atlas began publication in 1949. The finished work will comprise 11 sections in 39 parts, 5 of which have been already noted in this *Journal*. The plan of the work is to have specialists prepare their parts, and then they are issued as they become available. The material is confined to tumors, primary and secondary, of organs or regions of the body. It is intended to give special pathology rather than general. The plates are accompanied by a descriptive text outlining the description, definition, natural history, morphology, differential criteria and selected bibliography of each tumor described. The plates are fine, and the publishing is excellent. The price per part is very low. The set is recommended for all collections on pathology.

An Atlas of Surgical Exposures of the Extremities. By Sam W. Banks, M.D., associate professor of orthopedic surgery, Northwestern University Medical School, and attending orthopedic surgeon, Chicago Memorial Hospital and Woodlawn Hospital; and Harold Laufman, M.D., Ph.D., associate professor of surgery and director of experimental surgery, Northwestern University Medical School, and associate attending surgeon, Michael Reese Hospital. 4°, cloth, 391 pp., with 552 illustrations. Philadelphia: W. B. Saunders Company, 1953. $15.00.

This atlas presents a comprehensive coverage of surgery of all the extremities and their regions. Text accompanies all the plates. There is a good index and the publishing is excellent in every way. The illustrations are particularly fine. The book should prove valuable to surgeons and orthopedists.

Proceedings of the Annual Meeting: Council for High Blood Pressure Research, American Heart Association. 8°, paper, 113 pp., with illustrations. New York: American Heart Association, 1953. $1.75.

Twelve papers are published in this volume of proceedings: 6 scientific reports and 6 reports to the public. This last series tells about the Council and what it is, and discusses hardening of the arteries, coronary thrombosis and high blood pressure. There is also a report by Dr. Howard B. Sprague, of Boston, on rheumatic and congenital heart disease and heart surgery.

The Roentgen Aspects of the Papilla and Ampulla of Vater. By Maxwell H. Poppel, M.D., professor of radiology, New York University Post-Graduate Medical School, director of the Roentgen Ray Department, Bellevue Hospital, consultant in radiology, Veterans Administration Hospital, Bronx, New York, consulting radiologist, United States Naval Hospital, St. Albans, Long Island, and roentgenologist, New York University Hospital; Harold G. Jacobson, M.D., director, Department of Roentgenology, Hospital for Special Surgery, associate clinical professor of radiology, New York University Post-Graduate Medical School, attendant in radiology, United States Veterans Administration Hospital; and Robert W. Smith, M.D., senior resident in radiology, Veterans Administration Hospital, Bronx, New York. 8°, cloth, 195 pp., with 106 illustrations and 8 tables. Springfield, Illinois: Charles C Thomas, 1953. $8.50.

This monograph presents a complete roentgenologic survey of the anatomy, physiology and pathology of the region of the papilla and ampulla of Vater. There is a bibliography of 8 pages and a good index. The publishing is excellent in every way. The book should be in all collections on roentgenology and the digestive system.

Hospital Service in the United States: The 1952 census of hospitals. Thirty-Second Presentation of Hospital Statistics by the Council on Medical Education and Hospitals of the American Medical Association. Approved Schools for Medical Technologists, X-Ray Technicians, Physical Therapists, Occupational Therapists and Medical Record Librarians. 4°, paper, 103 pp. Chicago: Reprinted from the *Journal of the American Medical Association*, Hospital Number, 1953, with supplement.

Zeitloses und Zeitgebundenes in der Chirurgie. By Prof. Dr. Rudolf Nissen, director, Chirurgischen Universitätsklinik, Basel. 8°, paper, 32 pp. Stuttgart: Georg Thieme, 1953. DM 2.- (Distributed by Grune and Stratton, New York.)

Diabetic Manual for the Doctor and Patient. By Elliott P. Joslin, M.D., Sc.D., medical director, George F. Baker Clinic, New England Deaconess Hospital, and consulting physician, Boston City Hospital. Ninth edition. 16°, cloth, 315 pp., with 55 illustrations and 33 tables. Philadelphia: Lea and Febiger, 1953. $3.00.

Expert Committee on Insecticides: Fourth report. 8°, paper, 98 pp. Geneva: World Health Organization, 1952. $0.65. (World Health Organization, Technical Report Series, No. 54.)

Psychology and Alchemy. By C. G. Jung. Translated by R. F. C. Hull. 8°, cloth, 563 pp., with 270 illustrations. New York: Pantheon Books, 1953. $5.00. (Bollingen Series XX.)

The Revival of Interest in the Dream: A critical study of post-freudian psychoanalytic contributions. With an article on the "spoken word." Robert Fliess, M.D. 8°, cloth, 164 pp. New York: International Universities Press, 1953. $3.00.

Symptom und Kausalität: Grundfragen der Neurologie und Psychiatrie. By F. Llavero. With foreword by Prof. Dr. H. Pette. 8°, cloth, 251 pp, with 6 illustrations. Stuttgart: Georg Thieme, 1953. DM 33.-

Diseases of Muscle: A study in pathology. By Raymond D. Adams, M.A., M.D., associate clinical professor of neurology, Harvard Medical School, and chief, Neurological Service, Massachusetts General Hospital; D. Denny-Brown, M.D., D. Phil., F.R.C.P., James Jackson Putnam Professor of Neurology, Harvard Medical School, neurologist-in-chief and director, Neurological Unit, Boston City Hospital; and Carl M. Pearson, M. D. 8°, cloth, 556 pp., with 347 illustrations. New York: Paul B. Hoeber, 1953. $16.00.

NONCLINICAL NOTES

TRUSTEE OF BOSTON UNIVERSITY

Dr. J. Mark Hiebert, executive vice-president of Sterling Drug, Incorporated, has been elected to the Board of Trustees of Boston University for a term ending in 1958.

Dr. Hiebert received his degree from Boston University School of Medicine in 1932 and was formerly on the staffs of the Massachusetts General and Massachusetts Memorial hospitals.

NOTICES

SOUTH END MEDICAL CLUB

The December meeting of the South End Medical Club will be held at the headquarters of the Boston Tuberculosis Association, 554 Columbus Avenue, on Tuesday, December 15, at noon. The speaker will be Dr. Theodore B. Bayles, whose topic will be "Treatment of Rheumatoid Arthritis." All physicians are cordially invited.

UNITED STATES ATOMIC ENERGY COMMISSION FELLOWSHIPS

Eight fellowships in industrial medicine will be offered by the United States Atomic Energy Commission for 1954-1955. The program is designed to provide advanced training and experience for physicians, particularly in relation to atomic energy in industry. The fellowships are open to United States citizens (men or women) who hold medical degrees from approved schools, and who have had one year of internship.

Awards are for one year of academic training at institutions offering approved graduate courses in industrial medicine and related problems in health associated with the atomic-energy program. A second year of in-plant training may be applied for. The salary for the first year is $3600, with $350 additional for wife and each dependent child. (Tuition and laboratory fees will be paid.) The salary for the second year is $6000, with nothing additional.

Applications for fellowships should be submitted before January 1, 1954, to Dr. Henry A. Blair, AEC Fellowships in Industrial Medicine, Atomic Energy Project, University of Rochester, School of Medicine and Dentistry, Rochester, New York.

SOCIETY MEETINGS AND CONFERENCES

DECEMBER 5-10. American Academy of Dermatology and Syphilology. Page 914, issue of November 26.

DECEMBER 6. American Academy of Dental Medicine. Page 170, issue of July 23.

DECEMBER 8. New York Society for Circulatory Diseases. Page 870, issue of November 19.

DECEMBER 8 and 15. Boston City Hospital House Officers' Association Lectures. Page 798, issue of November 5.

DECEMBER 11 and 12. New York and Brooklyn Regional Committee on Trauma, American College of Surgeons. Page 828, issue of November 12.

DECEMBER 11 and 12. University of Buffalo Scientific Symposium. Page 8.0, issue November 19.

DECEMBER 11 and 12. Association of Research in Nervous and Mental Diseases. Further information may be obtained from Dr. Clarence C. Hare, Secretary, 710 West 168th Street, New York 32, New York.

DECEMBER 15. South End Medical Club. Notice above.

DECEMBER 18. Boston Lying-in Hospital Journal Club. Page 672, issue of October 15.

DECEMBER 21. Hartford Medical Society Program. Page 260, issue of Aug st 6.

DECEMBER 28. American Association for the Advancement of Science. Page 828, issue of November 12.

DECEMBER 28-30. American Society of Human Genetics. Page 914, issue of November 26.

JANUARY 4. American Federation for Clinical Research, Eastern Sectional Meeting. Jimmy Fund Building, 35 Binney Street, Boston.

JANUARY 6, MARCH 3 and April 21. Massachusetts Society for Research in Psychiatry. Page 750, issue of October 29.

JANUARY 22, 23, 29 and 30. International College of Surgeons Regional Meeting. Page 828, issue of November 12.

FEBRUARY 2-4. Examination for Public Health Service Medical Officers. Page 750, issue of October 29.

FEBRUARY 5 and APRIL 9. New England Society of Anesthesiologists. Page 914, issue of November 26.

FEBRUARY 15-20. Pan-American Academy of General Practice. Page 790, issue of November 5.

MAY 31-JUNE 3. American Urological Association. Page xvii, issue of July 23.

JULY 22 and 23. International College of Surgeons Regional Meeting. Page 828, issue of November 12.

JULY 23-29. International Cancer Congress. Page 914, issue of November 26.

SEPTEMBER 7-10. Nineteenth Annual Congress of the United States and Canadian Sections of the International College of Surgeons. Page 828, issue of November 12.

SEPTEMBER 12-25. Second International Congress of Cardiology. Page 44, issue of July 2.

SEPTEMBER 16-18. American Heart Association Annual Scientific Sessions. Page 44, issue of July 2.

DISTRICT MEDICAL SOCIETIES

ESSEX NORTH

JANUARY 13. Pentucket Club, 111 Main Street, Haverhill.

MAY 12. 113th Annual Meeting. Andover Country Club, Andover.

MIDDLESEX EAST

JANUARY 20. (Combined with the Auxiliary.)

MARCH 24.

MAY 11. Annual Meeting. (Harlow Dinner.)

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING THURSDAY, DECEMBER 10

THURSDAY, DECEMBER 10

*8:00-8:45 a.m. Case Presentations. Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.

*8:30-10:00 a.m. Orthopedic Staff Conference. Boston City Hospital.

*9:00-10:00 a.m. Arthritis Grand Rounds. Robert Breck Brigham Hospital.

10:30-11:15 a.m. Lecture on Diabetes for Doctors and Patients by a Member of the Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.

*11:00 a.m.-12:00 m. Pediatric Conference. Mount Auburn Hospital, Cambridge.

*11:00 a.m.-12:30 p.m. Hand Clinic (Physical Medicine and Rehabilitation Service). Boston City Hospital.

4:00 p.m. Combined Seminar Cancer Research Institute. Problems of Ion Transport. Dr. Frederick Snell. Children's Cancer Foundation. Conference Room 414, 35 Binney Street.

*4:00-5:00 p.m. Psychosomatic Diseases: Plan of Differential Diagnosis and Treatment. Dr. J. Bragan. Nurses' Home, Cambridge City Hospital.

5:00 p.m. Case Presentation and Pathological Discussion followed by a Subject of Special Presentation. Joslin Amphitheater, New England Deaconess Hospital.

*5:00-6:00 p.m. Surgical Division Meeting. Mount Auburn Hospital, Cambridge.

FRIDAY, DECEMBER 11

*8:00-8:45 a.m. Case Presentations. Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.

*9:30-11:30 a.m. Medical and Surgical Grand Rounds. Drs. George W. Thorn and Francis D. Moore. Main Amphitheater, Peter Bent Brigham Hospital.

*10:00 a.m. Tuberculosis Surgical Clinic. South End Health Unit, 57 East Concord Street.

10:30-11:15 a.m. Lecture on Diabetes for Doctors and Patients by a Member of the Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.

(Notices concluded on page xvii)